POST TRAUMATIC SEIZURE:

A PROSPECTIVE STUDY

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Dedicated

to

My Family

&

Teachers

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Abbreviations

1.	CBF	Cerebral blood flow
2.	CDC	Centre for disease control
3.	CT	Computed Tomography
4.	DAMA	Discharged against medical advice
5.	DBP	Diastolic blood pressure
6.	EDH	Extradural hemorrhage
7.	GCS	Glasgow coma scale
8.	GOS	Glasgow outcome scale
9.	Hb	Haemoglobin
10.	ICH	Intracerebral hematoma
11.	ICP	Intracranial pressure
12.	IVH	Intraventricular Haemorrhage
13.	MLS	Midline shift
14.	PTE	POST TRAUMATIC EPILEPSY
15.	PTS	Post Traumatic Seizure
16.	RL	Reacting to light
17.	RTA	Road traffic accident
18.	SAH	Subarachnoid Haemorrhage
19.	SDH	Subdural hematoma
20.	TBI	Traumatic brain injury

ABSTRACT

Introduction: Post-traumatic seizures (PTS) are a well-recognized sequela of traumatic brain. PTS have been documented as a common complication of TBI for decades. Incidence of PTS varies drastically throughout the literature and is dependent on many factors including study design and characteristics of the study population. As incidence of TBI increases and death due to TBI decreases, more individuals will be at risk of developing and living with chronic complications.

Aims and Objectives: To determine the frequency, time to event, and risk factors for posttraumatic seizures (PTS) following traumatic brain injury (TBI).

Material and methods: To determine the frequency, time to event, and risk factors for posttraumatic seizures (PTS) following traumatic brain injury (TBI) studied in 450 patients admitted in Dayanand medical college and hospital from 1st April 2019 to 31st May 2020.

Results: This study concluded that 450 post traumatic patients were taken, out of which 36(8 %) developed seizures. Of the total of 36 patients detected to have hemorrhagic contusion on CT scan, 12 patients developed seizures. Of the total of 70 patients detected to have depressed fracture on CT scan, seven developed seizures (10%). we found that the independent risk factors associated with occurrence of PTS were frontal—temporal lobar contusion, linear fracture and severity of head injury. All these findings were statistically significant.

Conclusion: we found that the independent risk factors associated with occurrence of PTS were frontal—temporal lobar contusion, linear fracture and severity of head injury. Thorough follow-up of patients with PTS and antiepileptic therapy may attain rehabilitation goals. Type of management (operative vs non-operative) does not affect the outcome of PTS.

Keywords: Traumatic brain injury, Glasgow coma scale, Elderly, Post traumatic seizure

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INTRODUCTION

Traumatic brain injury (TBI) is a significant public health concern and is a major cause of morbidity and mortality, especially to those under age 45years.[1,2] TBI is extremely heterogeneous regarding mechanism of injury, injury severity, and possible outcome. Individuals may experience many different complications and comorbidities associated with TBI that may continue chronically, persisting for many years or even throughout the lifetime following injury.[3-5]

Individuals with more severe injury are particularly affected by chronic conditions associated with TBI. Among individuals who survive a severe TBI, disability rates are estimated as high as 77%.[6] Chronic complications include, but are not limited to, poor functional outcome, decreased cognitive function, psychosocial and/or behavioral problems, decreased general health, and other neurological sequelae. These complications contribute substantially to the cost of care associated with TBI, which is estimated at more than $60 billion annually.[7] In addition to medical cost incurred, TBI may significantly increase years of potential life lost and decrease quality of life.

Post-traumatic seizures (PTS) are a well-recognized sequela of traumatic brain. PTS have been documented as a common complication of TBI for decades. Incidence of PTS varies drastically throughout the literature and is dependent on many factors including study design and characteristics of the study population. As incidence of TBI increases and death due to TBI decreases, more individuals will be at risk of developing and living with chronic complications. It can also be expected that PTS incidence will increase. Previously, seizure prophylaxis has been shown to be effective in reducing the incidence of PTS in the first week after injury, but has no long-term benefit (Temkin, 1990). Current recommendations from the American Academy of Neurology and the Brain Trauma Foundation include delivery of phenytoin for seizure prophylaxis during the first seven days post-TBI.[8,9] Yet, despite decades of research, there are no effective

pharmacological interventions to prevent post-traumatic seizures that develop after seven days post-injury and, it does not appear that rates of PTS are decreasing. Increasingly, novel risk factors for PTS and mechanisms of epileptogenesis following TBI are being investigated to identify potential new targets for therapeutic treatment.

1.1 TRAUMATIC BRAIN INJURY

1.1.1 Definitions

Traumatic brain injury is caused by an impact to the head from an external force that disrupts physiological function. External forces can include direct mechanical impact (i.e. blunt trauma), acceleration or deceleration associated injury (i.e. whiplash), blast injury caused by a pressure wave (i.e. explosion), or penetrating injury (i.e. gunshot).

Severity of TBI can be classified multiple ways. Originally developed to classify levels of consciousness [10], the Glasgow Coma Scale (GCS) has become the most widely used tool to describe TBI severity. The GCS score ranges from 3 to 15: 3-8 indicating severe, 9-12 indicating moderate, and 13-15 indicating mild TBI.[11] Alternative criteria based on loss of consciousness and post-traumatic amnesia are also used.[12] Centers for Disease Control and Prevention (CDC), continue to utilize a combination of clinical variables to assess TBI severity (Table 1).

Table 1. Classification of Traumatic Brain Injury

Criteria	Mild	Moderate	Severe
Structural Imaging[1]	Normal	Normal or abnormal	Normal or abnormal
Loss of Consciousness	0 - 30 min	> 30 min but < 24 hours	> 24 hours
Alteration ofConsciousness[2]	0 - 24 hours	> 24 hours	
Post-Traumatic Amnesia	≤ 24 hours	24 hours – 7 days	> 7 days
Glasgow Coma Scale[3]	13–15	9–12	3 – 8

[1]Abnormalities not related to trauma may be present with mild injury;
[2]Classification of severe injury not made based on alteration of consciousness alone;[3]Typically based on best GCS score is first 24 hours post-injury**adapted from Silver, McAllister, & Yudofsky. Textbook of Traumatic Brain Injury, Second Edition*

In addition to severity, TBI is often classified as closed-head or penetrating brain injury.

Many studies within the existing literature categorize TBI into one of four classifications: mild, moderate, severe, or penetrating. Despite the fact that it is possible to obtain a GCS score on an individual with a penetrating TBI (pTBI), researchers and clinicians often refer to pTBI as a distinct category.

As medical care has evolved, and computed topography (CT) became a part of standard care, neuroradiological findings have increasingly been used to differentiate and define TBI.

Abnormalities detected via CT are categorized based on location and type of injury [i.e. which hemisphere, brain region, and pathology present (Table 2)]. Advances in neuroradiological imaging through tools such as magnetic resonance imaging (MRI), positron emission topography (PET), and magnetic resonance spectroscopy (MRS), may provide additional ways to classify and differentiate brain injury. Some definitions regarding severity of TBI include positive evidence of abnormal pathology via CT imaging as a distinguishing characteristic.

Table 2. Abnormal Pathologies Identified via Computed Tomography

Variable	Definition
Intracranial Hemorrhage (ICH)	Bleeding within the skull (cranium)
Contusion	Area(s) of bleeding on the surface of the brain, most commonly along the undersurface and poles of the frontal and temporal lobes
Subdural Hematoma(SDH)	Presence of extra-axial blood clot or collection within sub-dural space, between surface of the brain and dura matter
Subarachnoid Hemorrhage (SAH)	Bleeding within the subarachnoid space, between the surface of the brain and arachnoid layer. May include blood in ambient, basal, interpenduncular cisterns or cisterna magna, or along falx or tentorium
Intra-ventricular Hemorrhage (IVH)	Blood documented within intra-ventricular space
Epidural Hematoma(EDH)	Presence of extra-axial collection within epidural space, between skulland dura matter
Penetrating TBI(pTBI)	Typically defined by penetration of the dura matter. May include bone,metal, or other foreign bodies present within the parenchyma, skull fractures displaced or depressed > 2mm, or “through and through” injuries penetrating the dura

The conceptual definition of epilepsy by the International League Against Epilepsy in 2005 is “a disorder characterized by an enduring pre-disposition to generate epileptic seizures and by neurobiological, cognitive, psychological and social consequences of this condition. The definition requires the occurrence of at least one epileptic seizure”.[13] An epileptic seizure is defined as “a transient occurrence of signs and /or symptoms due to abnormal excessive or synchronous neuronal activity in the brain”.[13] The usual operational definition identifies epilepsy after two or more unprovoked seizures occurring at least 24 hours apart.[14]

The latency of seizure occurrence is commonly categorized according to three intervals. Immediate seizures occur less than 24 hours after TBI, and early seizures occur less than one week after TBI, arising from the acute pathophysiologic changes in cerebral function immediately following TBI or in the acute period after TBI, respectively.[15] Late seizures occur more than a week after TBI, and if recurrent, constitute the diagnosis of PTE.[16] About 90% of seizures happening within the first four weeks after TBI will happen in the first week.[15]

The occurrence of early seizures is a recognized complication of TBI, and PTE is one of the major long-term outcomes of TBI.[15] The reported probability of developing epilepsy after TBI varies considerably.[12,16,17] In a study identifying 5984 episodes of TBI in Minnesota from 1935 to 1984, the probability of PTE ranges from 0.7% to 10.0% in five years follow-up and 2.1% to 16.7% in 30 years follow-up, correlating with the severity of TBI.[12] Other estimates of the incidence of epilepsy after TBI among civilian populations range from 2.1% to 16.3% for early seizures and 1.9% to 25.3% for PTE.[16] The cumulative incidence of epilepsy after severe penetrating TBI among military veterans is as high as 53%.[17]

Risk factors of PTE include chronic alcoholism, age of 65 years or older, penetrating injuries, intracranial hemorrhage, severity of injury,

posttraumatic amnesia or loss of consciousness for more than one day, focal neurologic deficits, depressed skull fractures, cerebral contusions, and retained bone and metal fragments.[12,18]

An important goal in the acute and long-term management of TBI is the prevention of PTE. Several randomized clinic trials have shown the effectiveness of anti-epileptic drugs (AEDs) in the management of early seizures after TBI.[19] Prophylactic anti-epileptics are effective in reducing early seizures after the head injury.[16,12] Longer, limited-term prophylactic use of three older AEDs: phenytoin (PHT), carbamazepine (CBZ), and valproate (VPA) have been evaluated in clinical trials, but have shown no benefit in the prevention of late seizures or epilepsy.[19,20]

AIMS & OBJECTIVES

To determine the frequency, time to event, and risk factors for posttraumatic seizures (PTS) following traumatic brain injury (TBI).

REVIEW OF LITERATURE

TBI is considered a “silent epidemic” because of a general lack of awareness of its magnitude in society.[21]

The estimated incidence of early seizures after TBI is between 2.6% and 16.3% depending on study populations (rehabilitation centers, or hospitalized).[22] Early seizures predispose to developing PTE, and are thought of as an epiphenomenon and result of an acute injury.[22] The pathophysiologic mechanisms leading to early seizures are not clearly understood, and multiple factors are thought to play a role, including interruption of the blood-brain barrier, presence of hemorrhage, and injury-related neuronal excitotoxicity.[22]

The overall incidence of PTE in hospitalized populations, comprising a range of TBI severity mainly from closed head injuries, is about 3-5%.[12,23] Military personels with penetrating head injury suffer the highest incidence of PTE, ranging from a 5-year cumulative incidence of 28% to a 15-year cumulative incidence of 53%.[17,24-26] In the series of non-missile combat injuries, that incidence reaches 12-24%.[23]

In about 6% of people with epilepsy in general populations, the condition is attributed to previous TBI.[22,27] The five-year cumulative probability of developing epilepsy was 0.7% following mild TBI, 1.2 % with moderate TBI, and 10% with severe TBI.[12] **Annegers et al** reported that the 30-year cumulative incidence was 2.1% with mild TBI, 4.2% with moderate TBI and 16.7% with severe TBI.[12] Compared to the uninjured population, the risk of PTE was increased 1.5 times after mild TBI, 2.9 times after moderate TBI and 17.0 times after severe TBI.[12]

TBI is one of the identified acquired causes of epilepsy. Added risk factors of PTE include chronic alcoholism, age of 65 years or older, penetrating injuries, intracranial hemorrhage, severity of injury, posttraumatic amnesia or loss of consciousness for more than one day,

focal neurologic deficits, depressed skull fractures, cerebral contusions, and retained bone and metal fragments.[12,18]

TBI is a multi-faceted disorder reflected in several potentially epileptogenic alterations in the brain. These include mechanical neuronal and vascular damage, parenchymal and subarachnoid hemorrhage, subsequent toxicity caused by iron-rich hemoglobin breakdown products, and energy disruption resulting in secondary injuries, including excitotoxicity, gliosis, and neuroinflammation.[28] It initiates cascades of molecular and cellular changes, resulting in several comorbidities, including epileptogenesis (the process by which a normal brain becomes epileptic, i.e., a process whereby CNS tissue acquires the capability to generate the abnormal and spontaneous electrical activity that underlies seizures) [29,30] and eventually PTE.[31] Brain injury, intracerebral hematoma formation, and hemorrhagic cerebral infarction cause extravasation of the intravascular contents, red blood cell (RBC) hemolysis, hemosiderin deposition within the neuropil and an increased incidence of epilepsy.[32] David Cantu *et al*. using controlled cortical impact in a rat model of TBI, found that specific cortical neuronal microcircuits may initiate and facilitate the spread of epileptiform activity following TBI.[33] They suggest increased glutamatergic signaling due to loss of GABAergic control may provide a mechanism by which TBI can give a rise to PTE.[33] PTE is the one of the most intractable consequences of TBI.[24] TBI may account for 10-20% of symptomatic epilepsy.[31] CT scan findings and neurosurgical procedures performed were the most useful findings in defining if individuals at high risk for PTE.[18]

Prophylaxis against early seizures is a part of standard therapy in the acute phase of moderate or severe TBIs, which are associated with acute increase in intra-cranial pressure.[22,34] Based on the guidelines from the Brain Trauma Foundation and the American Academic of Neurology, patients with moderate or severe TBI are typically placed on an AED right after the initial trauma, most commonly phenytoin (PHT), recently also levetiracetam (LEV), in order to prevent early

seizures.[22] If seizures are not present in the first 7 days after TBI, the AED is usually weaned, with an expectation that seizures in most instances will not occur in the future.[22] Information on the efficacy of using AEDs in the first 7 days after TBI in preventing the later development of PTE is sparse.

Reports are conflicting in regards of the efficacy of using AEDs in patients with TBI to prevent the development of PTE. PHT has been most frequently tested for an AED effect.[35,36] In 1979, a retrospective study showed that among 62 patients with severe TBI, 10% of the 50 treated with PHT and 50% of the 12 without PHT developed PTE, with a maximum follow-up period of six years.[36] But in 1983, randomized double-blind placebo-controlled studies showed that the early administration of PHT did not lessen the occurrence of early seizures[37], nor late seizures[38] after TBI. Another randomized double-blind placebo-controlled study with the largest sample size (408 patients) showed that there was a substantial effect of PHT on early seizures and lack of a positive effect on late seizures.[39] Then **Temkin *et al.*** reported a similar conclusion after a meta-analysis of all the trials.[40] **Willmore *et al.*** found that PHT treatment did not affect lipid peroxidation in rat with brain injury. They suggested treatment to prevent peroxidation may be more effective for epilepsy prophylaxis than using PHT, if PTE developed because of red blood cell extravasation, hemolysis, parenchymal deposition of heme compounds, and initiation of lipid peroxidation.[32]

In a 1983 study of 139 patients with severe TBI, the investigators started prophylaxis immediately after the accident and continued it for one and half to two years.[41] The results showed that carbamazepine (CBZ) reduced early seizures but did not affect the incidence of PTE even if seizures started during the treatment period.[42] A randomized, double-blind, single-center, parallel-group clinic trial on 379 patients with TBI was conducted in three groups: PHT for 1 week, or valproic acid (VPA) for 1 month, or VPA for 6-months. All the cases were followed up to 2 years.[23] The results showed VPA did

not offer any benefit for prevention of early or late seizures.[20] There was a trend toward a higher mortality rate among VPA-treated patients.[20] Formisano *et al.* found that there is no significant difference of development of PTE between TBI patients with and without prophylactic treatment.[34]

Thapa A and Chandra SP et al suggested that the risk of PTS was higher in patients who sustained fall from height, in GCS < 9, and associated medical problems. About 1/3rd of the patients with early PTS developed recurrent delayed seizures.[43]

However, there are theoretical grounds for the use of AEDs to prevent PTE.[22] Possibilities of effective AEDs include lamotrigine and topiramate, which exert activity against AMPA receptors.[22,44] Topiramate in the setting of therapeutic hypothermia shows neuroprotection.[45] In animal models, talampanel and parampanel, AMPA antagonists, and lacosamide show antiepileptogenic effect, and talampanel shows neuroprotective activity.[46,47] Another AMPA-receptor antagonist with similar action to parampanel, NS 1209 can decrease hyperexcitability by targeting glutamate activity at post-synaptic AMPA receptors and it also shows neuro-protection and effectiveness in epilepsy.[44]

Risk Factors of PTS

Few risk factors have been identified for PTS across all time-points (immediate, early, and late). Injury severity is the most commonly examined risk factor for PTS. Within the literature, there is general agreement that greater severity is associated with increased risk of PTS in all timeframes.[12,48]

Where there are slight differences in results regarding severity as a risk factor, the method used to categorize severity may be responsible. Englander and colleagues found the highest cumulative probability of late PTS among individuals with moderate TBI, classified by GCS (GCS 9-12), over the first two years post-injury.[18] However, this finding highlights the construct of survival bias, particularly for late

PTS. Individuals with severe TBI are more likely to expire within the first week post-injury, excluding them from being at risk for development of late PTS.

Further complicating the effect of injury severity on PTS, specifically immediate and early PTS, when continuous EEG monitoring is used to ascertain cases, there is no significant difference in injury severity, as measured by the GCS, between individuals who seize and those who do not.[49] As such, increasing injury severity may correlate with increasing risk of late PTS,limited to clinically apparent seizures. Using consistent metrics to classify injury and to ascertain PTS status would increase comparability of findings across studies.

Injury characteristics have been extensively studied as risk factors for PTS. As CT imaging became common practice following head trauma, specific pathology types were also examined. Presence of intra-cerebral blood, including intracerebral hemorrhage and subdural hematoma, has been identified as a risk factor for early and late PTS, increasing risk up to 30%.[12,18,48] While pTBI is often associated with late PTS, depressed skull fracture has been specifically documented as a risk factor for both early and late PTS.[12,18]

In addition to injury severity, age at injury is consistently cited as a risk factor for PTS. Children are at an increased risk of PTS at all time-points post-injury, but are particularly prone to immediate and early seizures.[12,50] The effect of age within adult populations is less clear. The Rochester Epidemiology Project found age greater than or equal to 65 was associated with a late PTS rate ratio of 2.5, which remained significant even after correction for other risk factors including early PTS and depressed skull fracture.[12]

Asikainen et al concluded increasing age among adults is correlated with increasing risk of late PTS.[51] Yet, other studies found no association between age and risk of late PTS.[52] Additional

research is needed to more thoroughly investigate the effect of age on late PTS among adults.

Early PTS

Characteristics of individuals who develop immediate and early PTS are more highly variable. In addition to risk factors for PTS during all timeframes discussed above, few risk factors are specific to immediate and/or early PTS. This is likely attributable to the fact that immediate and early are considered acute symptomatic seizures, direct responses to the head trauma. Therefore, research has not extensively examined risk factors for early PTS outside of injury severity. As more research is conducted, novel risk factors specific to immediate and/or early seizures may be identified.

Late PTS

Penetrating TBI is often defined by dura penetration and may include the presence of bone and/or foreign fragments (e.g. shrapnel). In both civilian and military cohorts, pTBI is one of the most prominent risk factors for late PTS.[18,51] **Salazar and Grafman et al** reviewed pTBI in both military and civilian cohorts and documented approximately 34 to 63% of individuals with pTBI develop late PTS.[53] Similarly, the highest probability of developing PTE in a civilian population, 62.5% in two years post-injury, was associated with bone or metal fragments and a relative risk of 3.94 compared to those with no dura penetration.[18]

Among individuals with late PTS, **Weiss and Caveness et al** found no statistically significant differences in seizure frequency for those with penetrating compared to non-penetrating TBI.[54] Interestingly, a penetrating wound greater than 3 cm deep was highly associated with increased seizure frequency [54], suggesting deep brain penetration may initiate different pathophysiological pathways leading to increased seizure.

As CT technology became increasingly available and used as part of the standard of care to diagnose TBI, abnormal neuroradiological findings were identified as risk factors for late PTS. Englander and colleagues found cisternal compression and midline shift, often associated with presence of intracranial bleeding and elevated intracranial pressure (ICP), were significantly associated with late PTS in a cohort of individuals hospitalized for moderate to severe TBI.[18] In addition to intracerebral blood collection (i.e. SDH, ICH), multiple studies have documented contusion as a significant risk factor for late PTS.[12,18,55] In a large population-based study, brain contusion and subdural hematoma remained significantly associated with late PTS after adjusting for additional risk factors including linear skull fracture, depressed skull fracture, and early seizure [rate ratios (95% CI); SDH: 6.3 (2.2-18.0), contusion: 5.0 (2.5-10.0)].[12] Risk of late PTS has also been shown to vary based on specific location of contusion (e.g. temporal) [18,56], likely related to certain brain regions/structures being more susceptible to seizure activity independent of TBI.

While few previous studies have specifically examined risk factors for early PTS, many studies document seizures occurring in the first week post-injury as a significant risk factor for late PTS. In univariate models, early seizures were shown to approximately double the probability of late PTS.[18,57] **Angeleri et al** determined the relative risk of late PTS was approximately 8.6 (95%CI: 2.9-25.6) for individuals with at least one early seizure compared to those with no early seizure activity.[56] Early seizures were also found to significantly increase risk for late PTS in multivariable models.[51,52] Contrary to these findings, early seizures were not determined to be significantly associated with late PTS in the Rochester Epidemiology Project cohort after controlling for other known risk factors.[12] Despite the majority of evidence indicating early seizures increase risk of late PTS, it remains unknown if immediate and early seizures have different effects on risk of lat PTS.

In a study conducted by **John F. Annegers et al**., 4541 patients were included in study of post-traumatic seizures for the 50-year period from 1935 through 1984. A total of 117 patients developed early post traumatic seizures. However the period of early seizure was extended to a month. Only 16 of the 117 patients who had early seizures had the first seizure more than one week after the injury. They identified that brain contusion and subdural hematoma were the strongest risk factors for late seizures. Skull fractures and prolonged loss of consciousness were significant but weaker predictors of late seizures.[12]

In a study conducted by **J. Englander et al.** a total of 647 subjects were enrolled during study period with the diagnosis of TBI, from August 1993 to November 1997 the number of subjects who experienced early posttraumatic seizure was 21 (3%). They studied the highest cumulative probability for late posttraumatic seizures in biparietal contusions. dural penetration with bone metal fragments. Multiple intracranial operations, multiple subcortical contusions, subdural hematoma with evacuation, midline shift greater the 5mm or multiple or bilateral cortical contusions.[18]

In a study conducted by **H. Wiedmeyer et al**. 1868 adult patients with head injury were analyzed retrospectively during a period of 11 years (from January 1998). In 109 patients (5.8%), an epileptic seizure occurred in the early post-traumatic period, 39 of these 109 patients with EPTS suffered from severe HI, 58 from moderate and 12 from mild The strongest single risk factor analysed was chronic alcohol abuse followed by subdural haematoma and brain contusion.[58]

In a study conducted by **S T Lee et al.**total of 2574 consecutively treated adult patients with moderate head injury were studied between July 084 to July 1994.There were 106 patients 01.1%) who developed early post traumatic seizures within first week of head injury.[59]

In a prospective study conduct by **S T Lee et al**., a total of 3340 consecutively treated adult patients with severe head injury were studied between July 1984 to July 1994. Each of them had a Glasgow Coma Scale (GCS) 3 to 8 after minimal Anticonvulsant agents were not given to these patients unless there was evidence of seizure. One hundred and twenty-one patients (3.6%) experienced seizures within I week after head injury; 42 of these (1.26%) had seizures within 24 hours after trauma.[59]

With advances in medicine and neuro-critical care, neurosurgical procedures have become common interventions following severe TBI. Neurosurgical procedures may include ventriculostomy, craniotomy, and craniectomy. Previous research indicates that neurosurgical interventions increase risk of late PTS.[57] Thus, its possible post-operative seizures within a certain timeframe may be considered acute symptomatic and not late PTS (i.e. PTE). Further research is needed to more comprehensively describe temporal trends of seizure activity post-surgery.

In addition to injury related characteristics, recent studies have investigated the effect of pre-morbid personal and medical history on late PTS. In a population-based sample of individuals hospitalized for TBI, pre-morbid history of depression [adjusted risk ratio (95% CI): 1.85 (1.16-2.94)] was significantly associated with increased risk for late PTS in multivariable analysis.[52] Although, seizures may also be caused by alcohol withdrawal and the duration of time between alcohol cessation and seizure development should be carefully inspected to ensure acute symptomatic seizures secondary to withdrawal[60] are not indicated as late PTS.

MATERIALS AND METHODS

SOURCE OF DATA

A prospective study was conducted on patients admitted with traumatic brain injury in Dayanand Medical College and Hospital, Ludhiana from 1st April 2019 to 31st May 2020.

METHODS OF COLLECTION OF DATA

Inclusion criterion

All patients presented with acute traumatic brain injury in Dayanand medical college and hospital Ludhiana was included in the study.

Exclusion criterion

- A preceding history of brain injury or major mental disorder,
- Pregnancy,
- Predisposition to seizures (such as a history of brain tumor or stroke),
- Family history of seizure

In this study, weused the classification of head injury severity as defined by Annegers et al. Mild injuries were those in which no skull fracture was found and the period of posttraumatic amnesia or loss of consciousness was ≤30 minutes. The moderately injured group of patients included those with skull fractures or other injuries with > 30 minutes of posttraumatic amnesia or loss of consciousness, who did not otherwise meet the criteria for a severe injury. Severe injuries were those with a documented brain contusion, an intracerebral hematoma (ICH) or > 24 hours of posttraumatic amnesia or loss of consciousness.[12]

Follow-up of 3 months was done

Variables like age, sex, trauma severity, GCS, onset of PTS, and neuroradiological findings, number of antiepileptic medications were studied.

Statistical Analysis

Data described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations were done

using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

OBSERVATIONS & RESULTS

Table 3: Post traumatic seizure in various age group.

		Post traumatic seizure				Total	Chi-square value	p-value
		N (n=414)		Y (n=36)				
Age GROUP	< 20	20	4.8%	2	5.6%	22	4.756	0.446
	21-30	122	29.5%	16	44.4%	138		
	31-40	80	19.3%	7	19.4%	87		
	41-50	85	20.5%	5	13.9%	90		
	51-60	53	12.8%	4	11.1%	57		
	> 60	54	13.0%	2	5.6%	56		

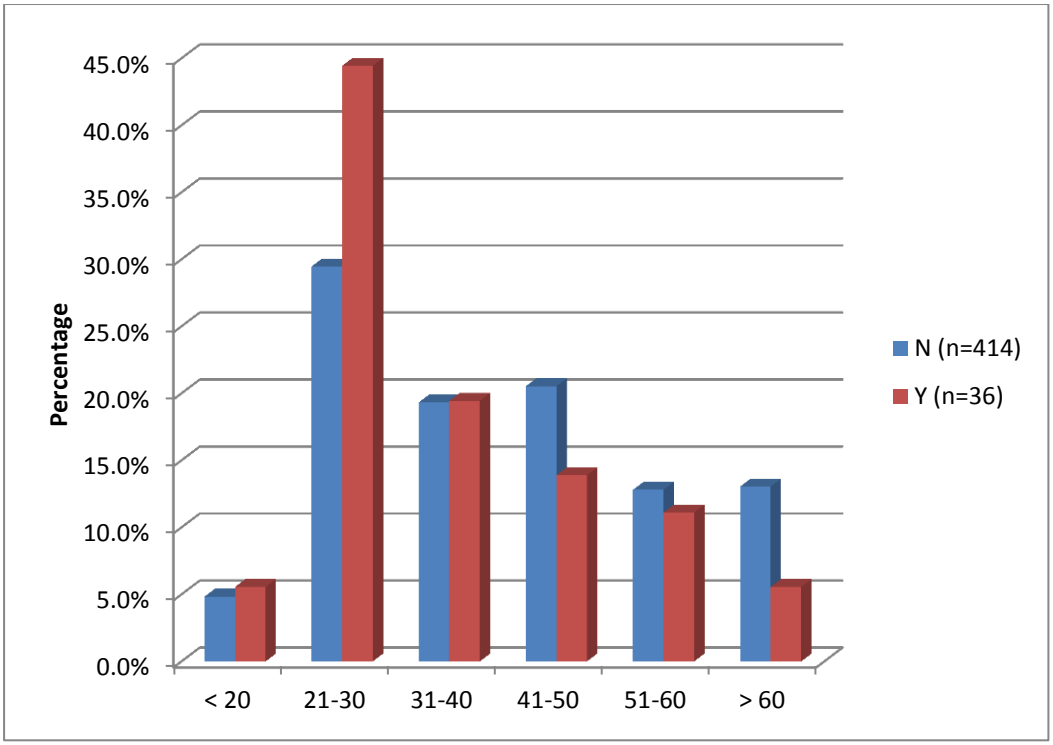

Figure 1: Post traumatic seizure in various age group.

Table 4: Post traumatic seizure in different gender.

		Post traumatic seizure				Total	Chi-square value	p-value
		N (n=414)		Y (n=36)				
Sex	F	81	19.6%	3	8.3%	84	2.752	0.119
	M	333	80.4%	33	91.7%	366		

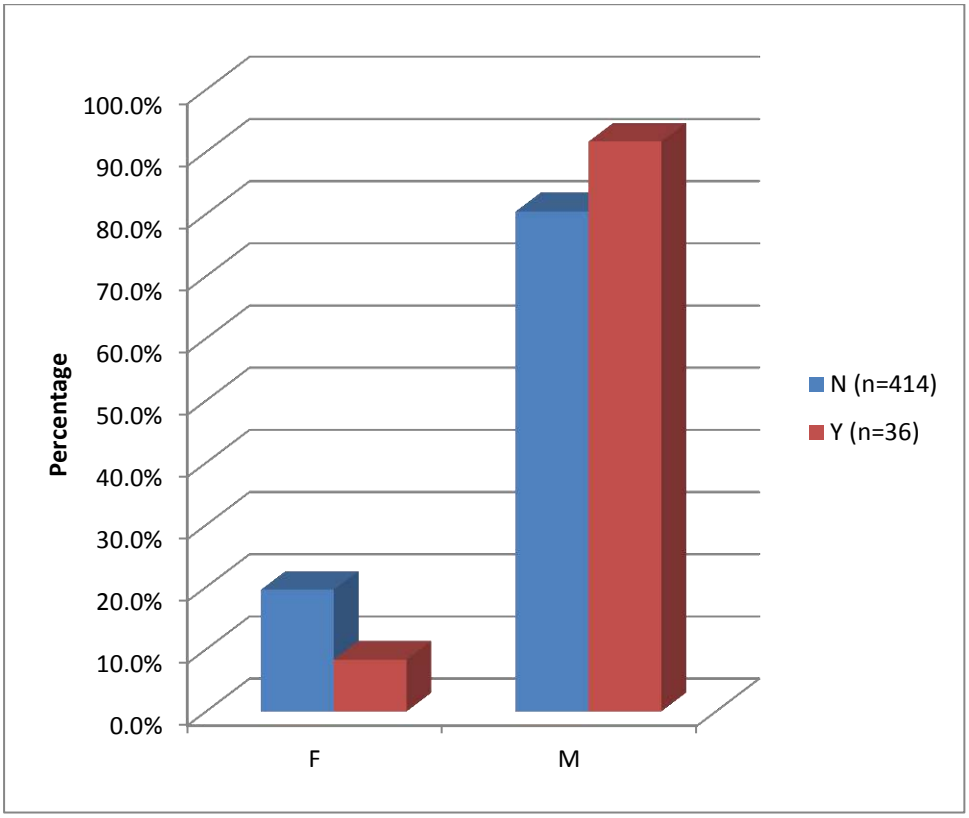

Figure 2: Post traumatic seizure in different gender.

Table 5: Post traumatic seizure in different types of injuries on CT.

		Post traumatic seizure				Total	Chi-square value	p-value
		N (n=414)		Y (n=36)				
IVH	N	397	95.9%	34	94.4%	431	0.172	0.678
	Y	17	4.1%	2	5.6%	19		
EDH	N	368	88.9%	35	97.2%	403	2.459	0.117
	Y	46	11.1%	1	2.8%	47		
SDH	N	292	70.5%	27	75.0%	319	0.320	0.571
	Y	122	29.5%	9	25.0%	131		
Cerebral hemoorhage	N	412	99.5%	36	100.0%	448	0.175	0.676
	Y	2	0.5%	0	0.0%	2		
Hemmoraha gic confusion	N	249	60.1%	24	66.7%	273	0.590	0.442
	Y	165	39.9%	12	33.3%	177		
SAH	N	347	83.8%	30	83.3%	377	0.006	0.940
	Y	55	13.3%	6	16.7%	61		
Intraparench ymal bleed	N	409	98.8%	34	94.4%	443	4.088	0.043
	Y	11	2.7%	2	5.6%	7		
Axonal injury	N	407	98.3%	35	97.2%	448	4.815	0.154
	Y	7	1.7%	1	2.8%	8		
Axial collection	N	402	97.1%	35	97.2%	437	0.002	0.967
	Y	12	2.9%	1	2.8%	13		
Suligalicalhe motoma	N	411	99.3%	33	91.7%	444	14.575	0.005
	Y	3	0.7%	3	8.3%	6		
Fracture	N	351	84.8%	29	80.6%	380	0.451	0.502
	Y	63	15.2%	7	19.4%	70		
Tentorium brush	N	413	99.8%	36	100.0%	449	0.087	0.768
	Y	1	0.2%	0	0.0%	1		

Figure 3: Post traumatic seizure in different types of injuries on CT.

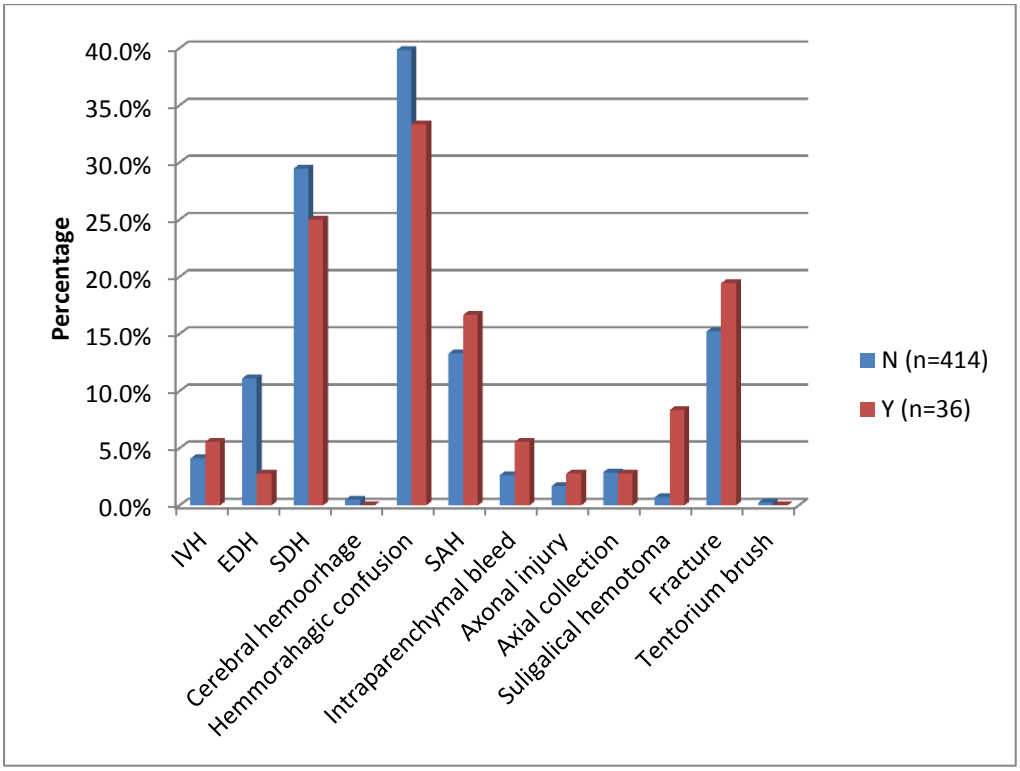

Table 6: Post traumatic seizure in various head injuries

		Post traumatic seizure				Total	Chi-square value	p-value
		N (n=414)		Y (n=36)				
Head Injury	Mild	260	62.8%	15	41.7%	275	0.654	0.844
	Moderate	69	16.7%	9	25.0%	78		
	Severe	85	20.5%	12	33.3%	97		

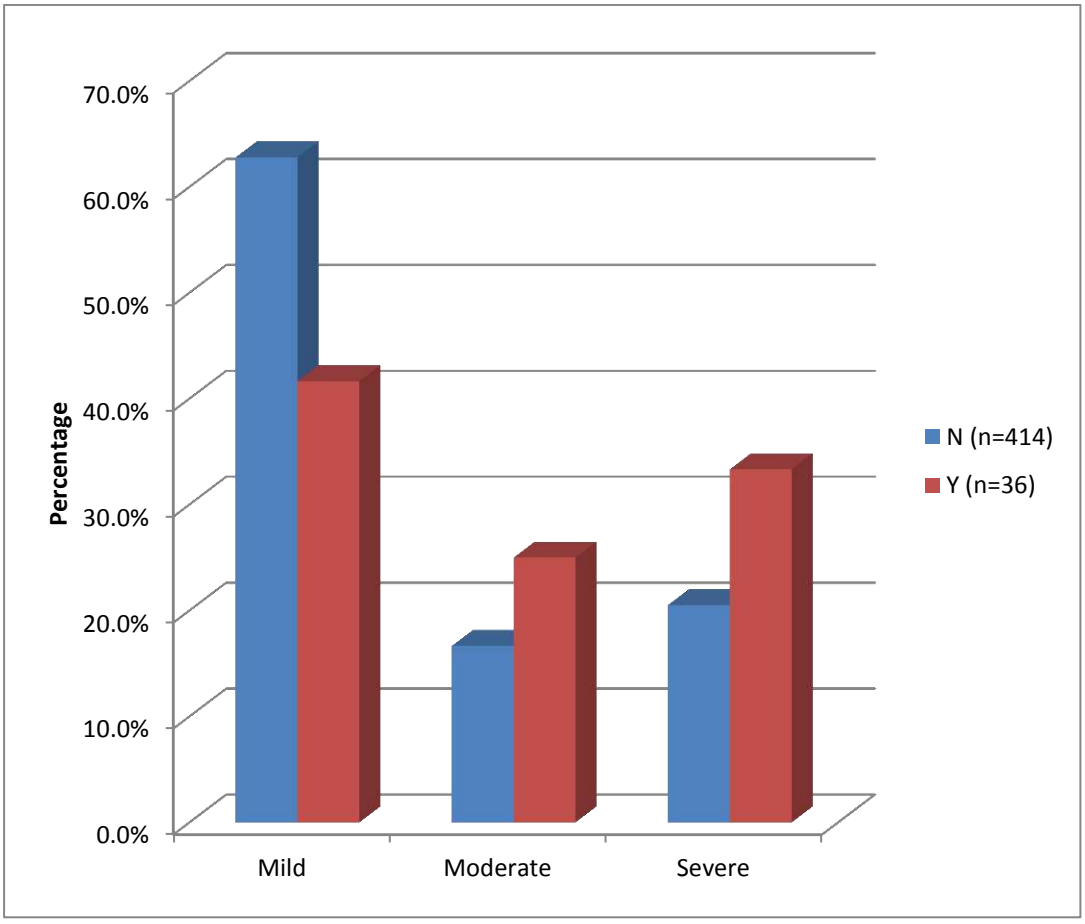

Figure 4: Post traumatic seizure in various head injuries

Table 7: Incidence of Post traumatic seizure in our study

		Post traumatic seizure				Total
		N (n=414)		Y (n=36)		
Complication	N	414	100.0%	36	100.0%	450

Table 8: Post traumatic seizure in Operative patients.

		Post traumatic seizure				Total	Chi-square value	p-value
		N (n=414)		Y (n=36)				
Type of Management S/NS	NS	250	60.4%	20	55.6%	270	0.322	0.57
	S	164	39.6%	16	44.4%	180		
Total		414	100.0%	36	100.0%	450		

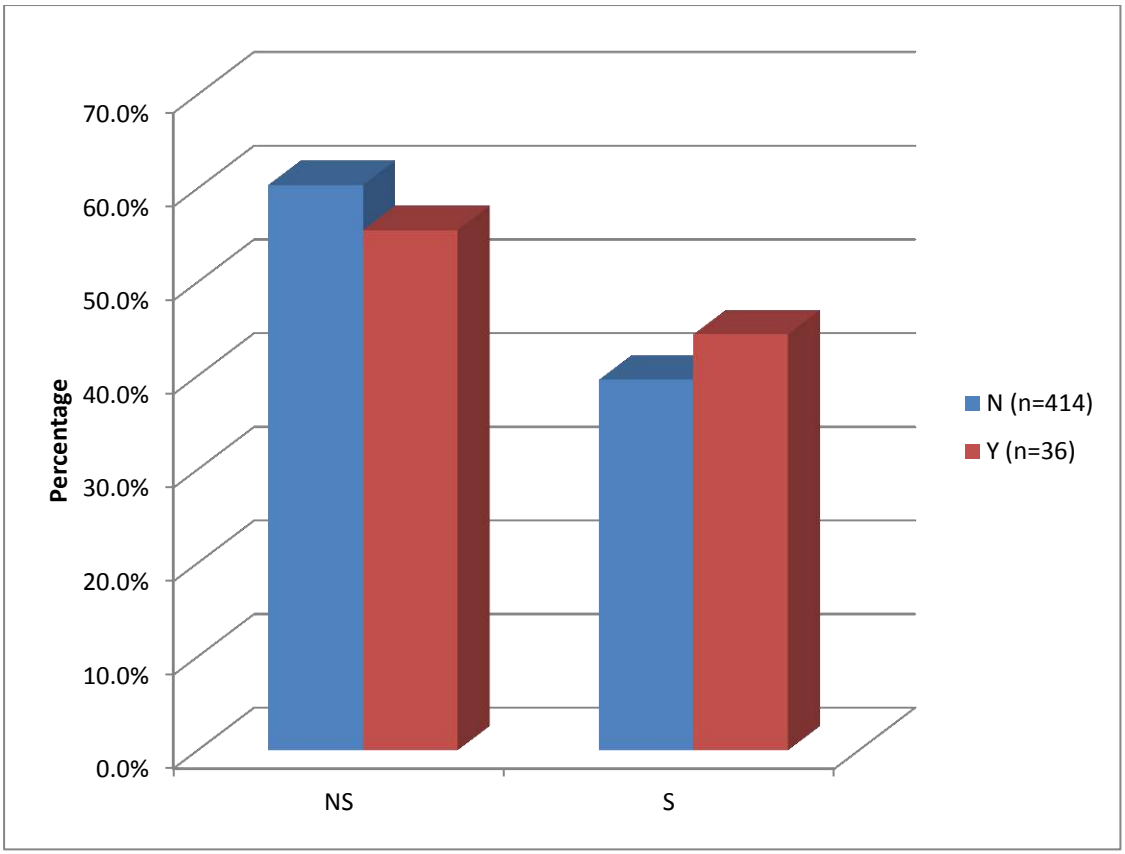

Figure 5: Post traumatic seizure in Operative patients.

Table 9: Mean and Standard deviation of Post traumatic seizure according to age, duration of stay and GCS.

	Post traumatic seizure	N	Mean	Std. Deviation	t	p-value
Age (In years)	N	414	41.00	15.462	1.826	0.069
	Y	36	36.14	13.784		
Duration of stay (In days)	N	414	17.04	19.444	0.002	0.998
	Y	36	17.03	20.948		
GCS	N	414	12.92	10.193	0.373	0.709
	Y	36	12.28	3.551		

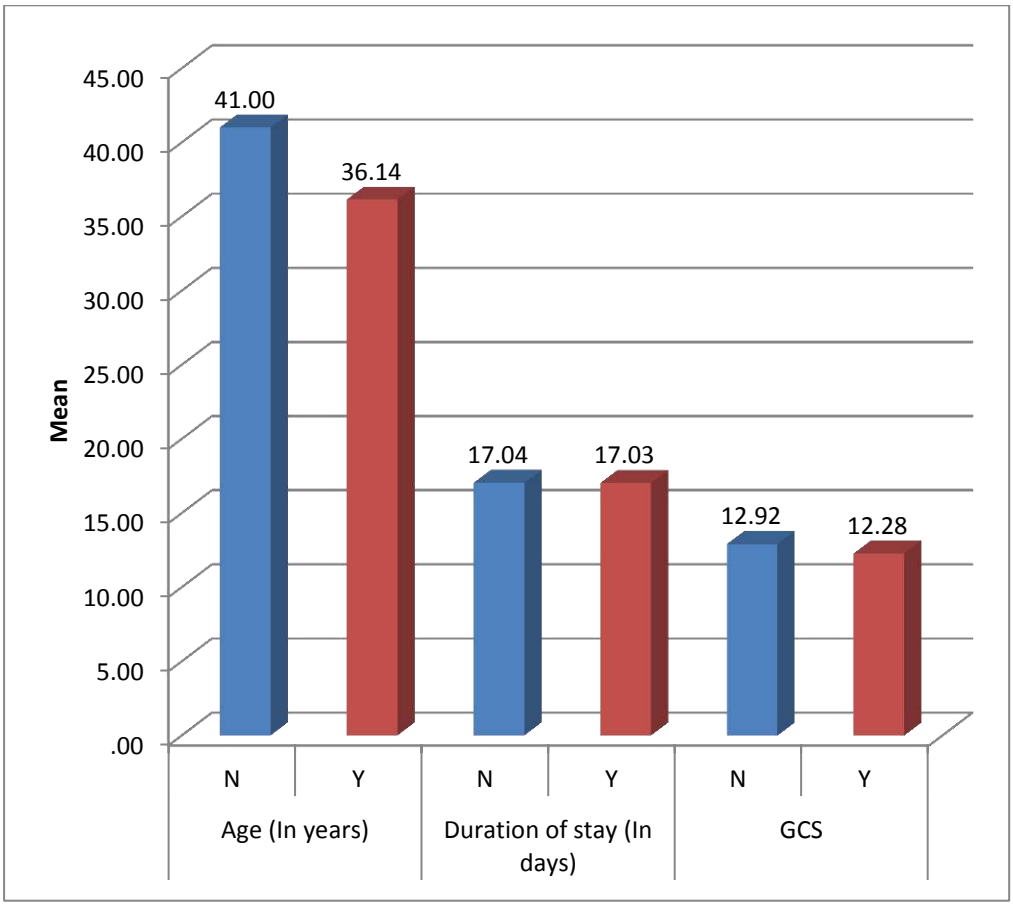

Figure 6: Mean and Standard deviation of Post traumatic seizure according to age, duration of stay and GCS.

A chi square test was performed for categorical data and mean & standard deviation were calculated for continuous data, further more parametric Pearson correlation test was tined for continuous data using SPSS version 17 (statistical package for social –(science) developed in year 2020. Assessment of Glasgow Coma Scale score with age was mated by the Pearson correlation coefficient (r). P-values were calculated using chi square for estimation of seizure status with the age (categorized) and mode of injury. Frequency table were also made for categorical data. A p value of less than 0.05 was considered significant.

In our study, 450 post traumatic patients were taken, out of which 36(8 %) developed seizures.

Patients with seizures were classified into three categories based on the Glasgow Coma Scale (GCS) score. The group with GCS score of 3-6 had 15 patients developing seizures out of 275 patients. The group with GCS score of 7-12 had 9 patients developing seizures out of 78 patients. The group with GCS score of 13-15 had 12 numbers of patients developing seiz ures out of 97 patients. Mean GCS of patients developing seizure is 12.28±3.551.

Patients were also classified on the basis of computerized tomography (CT) findings. Of the total of 36 patients detected to have hemorrhagic contusion on CT scan. 12 patients developed seizures (33%). Of the total of 8 patients diagnosed diffuse axonal injury, 1 patient developed seizures (12.5%). Of the total of 70 patients detected to have depressed fracture on CT scan, seven developed seizures (10%). Of the total of 47 patients diagnosed extra dural hemorrhage on CT scan 1 patient developed seizures (2%). Of the total of 61 patients diagnosed subarachnoid hemorrhage On C.T scan 6 patients developed seizures (9.8%), Of the total of 131 patients. detected to have subdural hemorrhage on CT scan, 9 patients developed seizures (6.8%). Of the 2 patients diagnosed intra parenchymal hemorrhage,

none developed seizures.13 patients had axial haemorrhage one developed seizure(7%).

Of the total 450 patients 270 were managed conservatively and 180 patients were operated. 20 patients out of 270 patients developed seizures. 164 patients were operated, 16 developed seizures.

In our study total number of patients were 450,22 patients below the age group of 20 out of which 2 developed seizure(5.6%). 138 patients in the age group of 21-30,out of which 16 developed seizures(44.4%). 87 patients were in the age group of 31-40,out of which 7 developed seizures (19.4%).90 patients were in the age group of 41-50 ,out of which 5 developed seizures(13.9).57 patients were in the age group of 51-60,out of which 4 developed seizures(11.1%). 56 patients had age more than 60 year ,out of which 2 developed seizures(5.6%). Mean age of patients developing seizure is 36.14±13.7.

In our study, out of 450 patients 366 were males and 84 were females.33 males and 3 females developed seizures. Mean duration of stay, in patients developing seizures is 20.948±0.002 years and in patient not developing seizure is 19.444±0.002 years.

DISCUSSION

The present study was carried out at Dayanand Medical College and Hospital, Ludhiana from 1st April 2019 to 31st May 2020 with the aim to determine the frequency, time to event, and risk factors for posttraumatic seizures (PTS) following traumatic brain injury (TBI).

During an 14-month period, from 1st April 2019 to 31st May 2020, 450 patients with diagnosis of traumatic brain injury (TBI) were evaluated. The patients underwent detailed clinical evaluation and radiological investigation as and when indicated. The documentation of a seizure was based on clinical findings alone either observed while in hospital or after discharge at home by a care giver.

In a study conducted by Shih-Tseng Lee et al. total of 4232 consecutively treated adult patients with mild closed head injury were examined. They ranged in age from 16 to 88 years mean(32.82 years); the male: female ratio was 3.8:1. The most common causes of trauma is motorcycle accidents (53.78%) and pedestrian injury (28.89%) In 100 patients (2.36) who developed seizures within the1st week after head injury, the male: female was 7:1 the mean age was 29.74 years.[59]

In this study subjects that were 21-30 Years at age were more likely to develop post traumatic seizures than subjects>30 years of age, but no significant difference between subjects 21-50 and those>50,years. Immaturity of the brain, which has been offered as the explanation for the grater susceptibility of children to early posttraumatic seizures, may explain the greater susceptibility of the younger age groups for seizures.

PTS is a familiar consequence of head injury. Many published documents have reported that. Identification of risk factors for PTS could facilitate reliable prediction of PTS and provide a theoretical basis for the prophylaxis of TBI patients. Different methods on collecting data lead to different incidence of PTS. It accounts for 5% of all patients in specialized epilepsy centers (Agrawal et al.,

2006).[60]Normally, the incidence in normal civilian is 1.8% to 5% (Frey, 2003; Jennett, 1974)[15,48] In our study, the overall incidence of PTS is 8%. In our study, we found that the independent risk factors associated with occurrence of PTS were frontal—temporal lobar contusion, linear fracture and severity of head injury measured using Glasgow coma scale (GCS). They all contributed to PTS. The frontal—temporal lobar contusion, parietal linear fracture and severity of head injury measured using Glasgow coma scale (GCS) were risk factors for PTS. Frontal—temporal lobar contusion is a risk factor for TBI patients in this study. Several reports (Annegers et al., 1998; Angeleri et al., 1999; Englander et al., 2003) have shown cerebral contusion was indeed a risk factor. Englander et al. (2003) reported the highest cumulative probability for late posttraumatic seizures included biparietal contusions (66%) and multiple or bilateral cortical contusions (25%).[12,18,56] Former studies reported that TBI has a predilection for frontal and temporal lobes. However, the mechanisms of PTS caused by contusion are still not clear. According to former studies (Willmore et al., 1978; Dakin and Weaver, 1993; Kharatishvili et al., 2006; Sharma et al., 2007), a possible explanation for this may be the following: cortical neurons injured following contusions, and this would lead the iron from the blood deposited in the cortex; the deposited iron would lead to iron-induced membrane peroxidation, and would make the seizure spread to the subcortical areas (hippocampus mainly); the occurrence and evolution of the epileptiform activity in the subcortical structures would in parallel with that in the cortical focus.[32,61-63] Further studies are needed to unveil this topic. The X-ray and CT findings showed that the linear fracture is significant in univariate analysis. In the multivariate model, it showed 2.966 times rate to have PTS compared to the no-finding group. This suggests people who had linear fracture were more prone to develop PTS. In a previous study, Annegers et al. (1998)[12] analyzed the linear fracture group and depressed fracture group individually firstly, and found the 2 groups had 2.2 and 1.8 times rate ratio, respectively, compared to the normal group. Then they combined the 2 factors together and found the rate ratio was 2.0. We found there

was no significance for the depressed fracture group in the unvariate model, while the linear fracture was a risk factor for PTS. This may be due to the individual characters of the patients. In our study, most of the linear fractures were parietal bone fracture, and some combined with cerebral contusion. The location and the combined clinical conditions may be able to explain why the linear fracture was a risk factor for PTS. More work need to be done to reveal this. TBI severity measured by GCS score is also a risk factor in this study. The patients with lower GCS scores (more severe injury) have more risk to develop PTS. Many documents (Haltiner et al., 1997[64]; Schütze et al., 1999[65]; Ferguson et al., 2010[52]) have reported that severity of the TBI patients was a risk factor for PTS and the risk was increased with the severity of TBI. In those studies, most of them used the GCS score to measure the brain severity; while a few (Annegers et al., 1998[12]) used other measurement (loss of consciousness or amnesia, skull fracture, subdural hematoma or brain contusion). All of those reported the risk of PTS increased with increased TBI severity despite different severity definitions. In our study, we used GCS score to measure the TBI severity and the results were consistent with the former studies. We found the patients with GCS scores from 3 to 8 had the highest seizure frequency (19.9%) and the highest risk to develop PTS (4.103 times risk rate). However, some studies showed the GCS score is not a risk factor. One study (Englander et al., 2003[18]) showed that GCS score alone could not predict late posttraumatic seizure risk . The authors found the patients whose GCS scores from 9 to 12 had the highest seizure frequency compared to those whose GCS scores from 3 to 8. The difference may be caused by different study methods, different entry criteria and different population the studies based and the characteristic of the patients. In our study, all the 3 groups of the patients (three severity levels) were recruited. The most severe injuries measured by GCS scores who had fatal injuries were excluded. The present study was carried on in the inpatients. Many persons with mild TBI seen in the emergency department were not admitted to the hospitals. All these factors may contribute to our findings and

differences observed. Different study methods, case selection and inclusion criteria make study comparisons different. In this study of TBI patients, frontal—temporal lobar contusion, linear fracture and severity of TBI measured by initial GCS were significant in the multivariate model analysis. Frontal—temporal lobar contusion, linear fracture and severity of TBI measured by initial GCS are risk factors for PTS. The reason PTS happens still needs to be investigated. Thorough follow-up of patients with PTS and antiepileptic therapy may attain rehabilitation goals. There is still a lot of work needed to be done.

SUMMARY & CONCLUSION

In conclusion, this study shows the epidemiologic features and risk factors for PTS after brain injury.

- In our study 8 % patients developed post traumatic seizures
- In our study, we found that the independent risk factors associated with occurrence of PTS were frontal—temporal lobar contusion and linear fracture
- Incidence of post traumatic seizure increased with more severity of head injury measured using Glasgow coma scale (GCS).
- Higher incidence of seizure seen in younger age group (21-30)

This study does provide a window to further investigate the risk factors for PTS in INDIAN population, and can help to set up an early warning system of critical illness for TBI patients in INDIA. In future studies, the prophylactic use of antiepilepsy drugs (AED) and rehabilitation of PTS patients must be expanded, combined with the individual characteristics, incidence and risk factors of PTS to provide TBI patients a better clinical diagnosis, treatment and rehabilitation.

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S no.	Name (Initials)	Age (In years)	Sex	MRD number	Duration of stay (In days)	GCS	IVH	EDH	SDH	Cerebral hemoorhage	Hemmorahagic confusion	SAH	Intraparenchymal bleed	Axonal injury	Axial collection	Suligalical hemotoma	Fracture	Tentorium brush	Head Injury	Post traumatic seizure	Complication	Type of Management S/NS
1	RK	30	M	882226	18	10	Y	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
2	KS	49	M	887175	11	9	N	Y	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
3	AS	25	M	851997	16	7	Y	Y	N	N	N	N	N	N	N	N	N	N	Severe	N	N	S
4	NS	22	M	885983	12	10	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	S
5	AC	50	M	891175	6	15	N	N	N	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
6	K	29	M	831731	114	8	N	N	N	N	N	N	Y	N	N	N	N	N	Severe	Y	N	S
7	AS	35	M	888796	12	6	N	N	N	N	N	Y	N	N	N	N	N	N	Severe	N	N	S
8	RR	60	M	869056	11	11	N	N	Y	N	N	N	N	N	N	N	N	N	Moderate	N	N	S
9	AS	34	M	889944	12	15	Y	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
10	NS	61	M	894118	6	15	N		N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
11	BS	24	M	895412	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
12	RS	26	M	895461	4	15	N	N	N	N	N	N	N	N	Y	N	N	N	Mild	N	N	NS
13	RK	20	M	889910	15	12	N	N	Y	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
14	VK	42	M	897102	3	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
15	T	36	M	859203	7	15	N	Y	N	N	N	Y	N	N	N	N	N	N	Mild	N	N	NS
16	SS	61	M	897132	6	15	N	N	N	N	N	N	N	N	N	N	N	Y	Mild	N	N	NS
17	SS	50	M	891202	15	15	N	N	N	N	Y	Y	N	N	N	N	N	N	Mild	N	N	S
18	SS	30	M	895658	2	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	Y	N	NS
19	BS	45	M	895671	10	13	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
20	RS	22	M	898037	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
21	SS	38	M	898419	9	15	N	Y	N	N	N	Y	N	N	N	N	N	N	Mild	N	N	NS
22	HK	50	F	845082	46	4	Y	N	Y	N	N	Y	N	N	N	N	N	N	Severe	N	N	S
23	JS	22	M	891168	23	6	N	N	Y	N	N	N	N	N	N	N	N	N	Severe	N	N	S
24	AS	22	M	897167	14	7	N	Y	N	N	N	N	N	N	N	N	N	N	Severe	N	N	S
25	CS	49	M	224265	31	13	N	N	Y	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
26	GS	68	M	890005	27	14	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
27	AS	42	M	901613	9	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
28	GS	52	M	895673	18	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
29	YM	55	M	899902	13	10	N	N	Y	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
30	GS	45	M	904175	10	13	N	N	Y	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
31	RK	55	M	904330	10	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
32	AM	20	M	899993	18	10	N	N	Y	N	N	N	N	N	Y	N	N	N	Mild	N	N	NS
33	MS	55	M	908472	9	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
34	RR	50	F	856766	43	6	N	N	Y	N	N	N	N	N	N	N	N	N	Severe	N	N	S
35	BD	30	M	904126	30	15	Y	N	Y	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
36	SS	32	M	912852	2	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
37	KS	21	M	898511	28	7	N	N	N	N	N	N	N	Y	N	N	N	N	Severe	Y	N	NS
38	RS	38	M	884738	51	5	N	N	N	N	N	N	N	N	N	N	Y	N	Severe	N	N	S
39	PK	46	F	815494	172	4	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
40	R	35	F	913038	7	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
41	VKV	67	M	282219	7	14	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
42	P	28	F	891154	47	7	N	N	N	N	N	N	N	N	N	N	N	N	Severe	N	N	NS
43	SK	65	F	915448	9	14	N	N	N	N	Y	Y	N	N	N	N	Y	N	Mild	N	N	NS
44	AS	50	M	910247	1	8	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
45	MK	37	M	919261	5	15	N	Y	N	N	N	Y	N	N	N	N	Y	N	Mild	N	N	NS
46	AS	28	M	919260	6	15	N	N	Y	N	N	Y	N	N	N	N	N	N	Mild	N	N	NS
47	SS	40	M	919971	5	15	N	N	Y	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
48	SK	34	M	904301	8	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
49	R	50	F	882214	13	8	N	N	N	N	N	N	N	N	N	N	N	N	Severe	N	N	NS
50	JS	65	M	887183	6	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
51	LS	20	M	877797	10	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
52	PK	20	M	880600	7	14	N	N	N	N	N	N	N	N	N	Y	N	N	Mild	N	N	NS

S no.	Name (Initials)	Age (In years)	Sex	MRD number	Duration of stay (In days)	GCS	IVH	EDH	SDH	Cerebral hemoorhage	Hemmorahagic confusion	SAH	Intraparenchymal bleed	Axonal injury	Axial collection	Suligalical hemotoma	Fracture	Tentorium brush	Head Injury	Post traumatic seizure	Complication	Type of Management S/NS
53	DS	27	M	877365	14	12	N	N	N	N	N	N	N	N	Y	N	N	N	Moderate	Y	N	S
54	KS	40	M	870578	30	5	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
55	IG	60	F	883316	1	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
56	RS	55	M	877986	14	13	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	S
57	GS	62	M	882156	5	14	N	N	N	N	N	Y	N	N	N	N	N	N	Mild	N	N	NS
58	BN	61	M	884762	2	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
59	VM	28	M	882205	10	6	N	N	N	N	N	N	N	N	N	N	N	N	Severe	N	N	S
60	GS	22	M	884045	8	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
61	DS	24	M	873601	31	4	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
62	PK	50	F	877987	13	8	N	N	Y	N	N	N	N	N	Y	N	N	N	Severe	N	N	S
63	RK	65	M	927561	7	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
64	PK	50	F	928212	35	15	N	N	N	N	N	Y	N	N	N	N	N	N	Mild	N	N	S
65	SS	28	M	924184	24	6	N	N	Y	N	N	Y	N	N	N	N	N	N	Severe	N	N	S
66	KK	37	F	925034	19	11	N	N	Y	N	Y	N	N	N	N	N	N	N	Moderate	N	N	S
67	HS	60	M	928062	2	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
68	AS	50	M	924147	22	12	N	N	Y	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
69	GS	38	M	412487	8	14	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
70	PK	64	M	922977	31	12	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
71	KB	38	M	916548	66	7	Y	Y	N	N	Y	Y	N	N	N	N	Y	N	Severe	N	N	NS
72	JK	48	F	924367	19	7	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
73	HS	23	M	890843	13	7	N	Y	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
74	N	65	F	921242	41	7	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
75	KS	48	M	924768	11	7	Y	Y	Y	N	N	N	N	N	N	N	N	N	Severe	Y	N	S
76	KR	57	M	924888	6	12	N	N	N	N	Y	Y	N	N	N	N	N	N	Mild	N	N	S
77	SS	22	M	781250	4	15	N	Y	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
78	M	24	M	698811	7	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
79	G	24	M	694812	16	7	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
80	S	41	M	6774229	12	15	N	N	N	N	N	Y	N	N	N	N	Y	N	Mild	N	N	NS
81	B	72	M	701104	13	7	N	N	Y	N	Y	Y	N	N	N	N	N	N	Severe	N	N	NS
82	R	50	M	704058	9	15	N	N	N	Y	N	N	N	N	N	N	N	N	Mild	N	N	NS
83	H	32	M	702792	10	15	N	N	N	N	N	N	N	N	Y	N	N	N	Mild	N	N	NS
84	G	40	M	701222	16	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
85	AK	45	F	701144	17	15	N	N	Y	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
86	J	25	M	705930	11	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
87	PS	40	M	704276	14	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
88	BS	35	M	711436	3	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
89	VK	50	F	710252	7	15	N	N	N	N	Y	N	N	N	N	N	Y	N	Mild	N	N	NS
90	S	30	M	672925	68	7	N	N	Y	N	Y	Y	N	N	N	N	Y	N	Severe	N	N	S
91	K	41	M	713054	4	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
92	P	41	F	704752	12	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
93	AS	60	M	714592	4	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	Y	N	NS
94	V	51	M	708707	14	11	N	Y	Y	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
95	N	17	F	716150	4	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
96	K	22	M	708504	13	12	N	N	Y	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
97	H	70	M	696293	35	4	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
98	NP	34	F	688090	46	6	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
99	A	20	M	702795	27	6	N	N	Y	N	Y	Y	N	N	N	N	N	N	Severe	N	N	S
100	SS	35	M	716187	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
101	S	35	M	717537	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
102	D	50	M	717703	15	11	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
103	RD	70	M	696524	42	7	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
104	MJ	38	M	720716	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
105	SS	70	M	707360	27	6	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS

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106	SK	58	M	704339	5	12	N	N	Y	N		N	N	N	N	N	N	N	Moderate	Y	N	NS
107	PS	25	M	716203	18	13	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
108	VK	65	M	717736	16	11	N	N	Y	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
109	PK	45	M	719182	16	12	N	N	N	N	N	N	Y	N	N	N	N	N	Moderate	N	N	S
110	MS	28	M	723985	10	14	N	N	Y	N	N	Y	N	N	N	N	N	N	Moderate	N	N	NS
111	MS	26	M	722174	13	13	N	N	Y	N	Y		N	N	N	N	N	N	Mild	N	N	NS
112	PS	55	M	717745	18	10	N	Y	N	N	N	Y	N	N	N	N	N	N	Moderate	N	N	S
113	BK	46	M	723944	10	12	N	N	N	N	Y	Y	N	N	N	N	N	N	Moderate	N	N	NS
114	GS	36	M	717781	20	6	Y	N	N	N	N	N	N	N	N	N	Y	N	Severe	N	N	S
115	RS	48	M	722076	16	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	Y	N	NS
116	GS	26	M	729411	6	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
117	JK	50	M	694884	59	6	N	N	N	N	N	Y	N	N	N	N	N	N	Severe	N	N	S
118	RS	35	M	698822	52	3	N	Y	Y	N	Y	Y	N	N	N	N	N	N	Severe	N	N	S
119	RK	29	M	723946	16	7	N	N	Y	N	N	Y	N	N	N	N	N	N	Severe	N	N	S
120	DR	65	F	729737	6	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
121	Y	15	F	730018	8	8	N	Y	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
122	G	45	M	733140	5	15	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
123	KS	42	M	734495	4	14	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
124	A	25	M	728763	10	9	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	S
125	AR	54	M	727011	15	13	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
126	C	35	M	731554	10	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
127	S	18	M	676628	18	7	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	Y	N	S
128	AK	27	M	733094	10	8	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
129	J	21	M	736119	10	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	S
130	KS	56	M	733216	15	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
131	R	26	F	731443	18	15	N	N	Y	N	N	N	N	N	N	N	N	N	MILD	N	N	S
132	HS	50	M	734518	13	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	S
133	A	29	M	740461	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
134	S	79	F	740618	5	14	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
135	NS	21	M	736153	11	12	N	N	N	N	N	N	N	N	N	N	Y	N	Moderate	N	N	S
136	PK	60	F	745044	4	150	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
137	HS	26	M	729478	27	13	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	Y	N	S
138	KK	59	F	565098	49	13	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
139	RS	32	M	722169	42	9	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
140	DS	26	M	747903	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
141	P	31	M	745913	5	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
142	RD	62	F	746627	7	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
143	S	50	M	710348	62	10	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	S
144	JS	70	M	745071	9	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
145	GS	30	M	745072	7	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	Y	N	S
146	HK	38	F	749085	7	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
147	GK	50	F	743413	16	14	N	N	N	N	N	N	Y	N	N	N	N	N	Mild	N	N	S
148	JS	29	M	752056	4	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
149	KS	21	M	749150	9	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
150	TS	83	M	751980	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
151	AK	38	M	733411	36	13	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
152	GS	61	M	753637	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
153	NS	24	M	756339	1	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
154	NS	56	M	756539	2	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	Y	N	S
155	RK	65	M	755208	4	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
156	RJ	18	M	749149	14	15	N	Y	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
157	MS	23	M	753463	7	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S

S no.	Name (Initials)	Age (In years)	Sex	MRD number	Duration of stay (In days)	GCS	IVH	EDH	SDH	Cerebral hemoorhage	Hemmorahagic confusion	SAH	Intraparenchymal bleed	Axonal injury	Axial collection	Suligalical hemotoma	Fracture	Tentorium brush	Head Injury	Post traumatic seizure	Complication	Type of Management S/NS
158	NK	40	M	757980	14	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
159	BS	45	M	757934	1	11	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
160	JS	45	M	763549	5	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	S
161	HR	58	M	756601	19	10	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
162	PC	60	M	565005	7	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
163	R	34	M	568525	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
164	M	40	M	704201	55	4	N	N	Y	N	N	N	N	N	N	N	N	N	Severe	Y	N	S
165	NR	58	M	756677	18	12	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	S
166	KS	24	M	767067	1	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
167	I	54	M	4559	2	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
168	AT	19	M	757981	18	10	N	N	N	N	N	N	N	N	N	N	Y	N	Moderate	N	N	NS
169	A	30	M	743508	41	5	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
170	PK	30	F	767169	3	15	N	N	N	N	N	Y	N	N	N	N	N	N	Mild	N	N	NS
171	RS	22	M	767030	6	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
172	AK	35	F	767040	6	12	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
173	SR	24	M	714803	88	4	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
174	RK	35	M	758089	25	8	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
175	SK	22	M	767241	8	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	Y	N	S
176	MS	55	M	766977	7	11	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
177	GS	45	M	767140	11	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
178	H	25	M	771757	6	10	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
179	KK	70	M	771768	6	15	N	N	Y	N		N	N	N	N	N	Y	N	Mild	N	N	NS
180	GS	21	M	772799	1	15	N	Y	N	N	Y	N	N	N	N	N	Y	N	Mild	N	N	NS
181	MK	50	F	782962	15	10	N	N	N	N	Y	Y	N	N	N	N	Y	N	Moderate	N	N	S
182	SR	24	M	714803	44	9	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	S
183	SS	31	M	793545	10	15	N	N	N	N	N	N	N	N	Y	N	N	N	Mild	N	N	NS
184	MS	45	M	798506	12	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
185	HS	28	M	802213	2	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
186	PS	28	M	793539	13	6	N	N	N	N	Y	N	N	N	N	N	Y	N	Severe	N	N	NS
187	AS	49	M	802202	3	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
188	MS	35	M	800354	7	13	N	N	N	N	N	Y	N	N	N	N	Y	N	Moderate	Y	N	NS
189	GS	45	M	779786	35	11	Y	N	Y	N	Y	N	N	N	N	N	Y	N	Mild	N	N	NS
190	KS	35	M	802244	6	14	N	N	N	N	Y	N	N	N	N	N	Y	N	Mild	N	N	S
191	S	65	F	805553	1	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
192	SS	31	M	799518	11	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
193	PS	25	M	805338	3	15	N	N	Y	N	Y	N	N	N	N	N	Y	N	Mild	N	N	NS
194	JS	29	M	800382	10	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
195	DB	26	M	805538	3	10	Y	N	N	N	Y	N	N	N	N	Y	N	N	Moderate	N	N	S
196	MS	21	M	776603	47	6	Y	N	N	N	Y	Y	N	N	N	N	Y	N	Severe	N	N	S
197	BS	32	M	787442	32	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
198	HS	45	M	807344	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
199	SK	40	F	810451	5	15	N	N	N	N	Y	N	N	N	N	N	Y	N	Mild	N	N	NS
200	AS	22	M	807305	9	15	N	N	N	N	Y	N	N	N	N	N	Y	N	Mild	Y	N	S
201	SK	27	F	783667	47	10	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
202	KP	46	F	783252	47	13	N	N	N	N	Y	N	N	N	N	N	Y	N	Mild	N	N	S
203	AS	30	M	811919	8	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
204	AA	18	M	807360	14	10	N	N	N	N	N	N	N	N	N	N	Y	N	Moderate	N	N	S
205	M	63	M	778340	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
206	JS	36	M	778384	4	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
207	PS	57	M	759780	34	11	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
208	MS	30	M	779759	11	13	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
209	AS	25	M	767075	31	13	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	Y	N	NS

S no.	Name (Initials)	Age (In years)	Sex	MRD number	Duration of stay (In days)	GCS	IVH	EDH	SDH	Cerebral hemoorhage	Hemmorahagic confusion	SAH	Intraparenchymal bleed	Axonal injury	Axial collection	Suligalical hemotoma	Fracture	Tentorium brush	Head Injury	Post traumatic seizure	Complication	Type of Management S/NS
210	RK	31	M	781238	10	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
211	M	55	F	781199	12	15	N	N	N	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
212	NS	52	M	762528	41	12	N	N	N	N	N	Y	N	N	N	N	N	N	Moderate	N	N	S
213	ML	28	F	756591	47	3	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
214	RK	57	M	786051	7	15	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
215	HK	45	F	786227	5	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
216	PK	26	F	786944	7	14	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
217	HS	55	M	776706	24	15	N	N	N	N	N	Y	N	N	N	N	N	N	Mild	N	N	S
218	AS	25	M	786224	9	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
219	MS	48	M	732479	90	15	N	N	Y	N	N	Y	N	N	N	N	N	N	Mild	N	N	S
220	HK	58	F	762449	45	15	N	N	Y	N	N	Y	N	N	N	N	N	N	Mild	N	N	S
221	H	25	M	790887	4	15	N	N	N	N	N	Y	N	N	N	N	Y	N	Mild	N	N	S
222	P	38	M	786245	9	15	N	N	N	N	N	N	N	N	N	Y	N	N	Mild	Y	N	S
223	JS	21	M	783078	14	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
224	JS	42	M	787506	7	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
225	AS	20	M	753477	60	5	N	N	N	N	Y	Y	N	N	N	N	N	N	Severe	N	N	NS
226	GS	24	M	786240	11	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
227	AK	35	F	790239	5	15	N	N	Y	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
228	JS	30	M	787485	12	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
229	B	30	M	756661	60	8	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
230	JK	21	F	791851	7	15	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
231	D	25	M	775097	34	15	N	N	Y	N	N	N	N	N	N	N	N	N	Moderate	N	N	S
232	G	20	M	795057	3	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
233	AS	50	M	778460	30	15	N	N	N	N	N	Y	Y	N	N	N	N	N	Mild	N	N	S
234	PK	25	F	793267	7	11	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
235	DS	40	M	793507	7	12	N	N	Y	N	Y	Y	N	N	N	N	Y	N	Mild	N	N	NS
236	MS	65	M	785978	19	8	N	N	Y	N	Y	N	N	N	N	N	N	N	Moderate	N	N	S
237	RS	27	M	796810	3	15	N	N	N	N	N	Y	N	N	N	Y	N	N	Mild	Y	N	NS
238	S	50	M	710348	70	11	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	S
239	BS	50	M	705699	36	8	N	N	N	N	N	N	N	N	N	N	Y	N	Moderate	N	N	S
240	AS	42	M	901613	9	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
241	GS	52	M	987623	18	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
242	YM	55	M	890004	13	10	N	N	Y	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
243	GS	45	M	901457	10	13	N	N	Y	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
244	RK	55	M	940033	10	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
245	AM	20	M	899994	18	10	N	N	Y	N	N	N	N	N	Y	N	N	N	Mild	N	N	NS
246	MS	55	M	904827	9	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
247	RR	50	F	857676	43	6	N	N	Y	N	N	N	N	N	N	N	N	N	Severe	N	N	S
248	BD	30	M	901425	30	15	Y	N	Y	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
249	SS	32	M	915825	2	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	Y	N	NS
250	KS	21	M	985225	28	7	N	N	N	N	N	N	N	Y	N	N	N	N	Severe	N	N	NS
251	RS	38	M	887438	51	5	N	N	N	N	N	N	N	N	N	N	Y	N	Severe	N	N	S
252	PK	46	F	814549	172	4	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
253	R	35	F	913038	7	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
254	VKV	67	M	282219	7	14	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
255	P	28	F	891154	47	7	N	N	N	N	N	N	N	N	N	N	N	N	Severe	N	N	NS
256	SK	65	F	915448	9	14	N	N	N	N	Y	Y	N	N	N	N	Y	N	Mild	N	N	NS
257	AS	50	M	910247	1	8	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
258	MK	37	M	919261	5	15	N	Y	N	N	N	Y	N	N	N	N	Y	N	Mild	N	N	NS
259	AS	28	M	919260	6	15	N	N	Y	N	N	Y	N	N	N	N	N	N	Mild	N	N	NS
260	SS	40	M	919971	5	15	N	N	Y	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
261	SK	34	M	904301	8	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS

S no.	Name (Initials)	Age (In years)	Sex	MRD number	Duration of stay (In days)	GCS	IVH	EDH	SDH	Cerebral hemoorhage	Hemmorahagic confusion	SAH	Intraparenchymal bleed	Axonal injury	Axial collection	Suligalical hemotoma	Fracture	Tentorium brush	Head Injury	Post traumatic seizure	Complication	Type of Management S/NS
262	R	50	F	882214	13	8	N	N	N	N	N	N	N	N	N	N	N	N	Severe	N	N	NS
263	JS	65	M	887183	6	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
264	LS	20	M	877797	10	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
265	PK	20	M	880600	7	14	N	N	N	N	N	N	N	N	N	Y	N	N	Mild	Y	N	NS
266	DS	27	M	877365	14	12	N	N	N	N	N	N	N	N	Y	N	N	N	Moderate	N	N	S
267	KS	40	M	870578	30	5	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
268	IG	60	F	883316	1	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
269	RS	55	M	877986	14	13	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	S
270	GS	62	M	882156	5	14	N	N	N	N	N	Y	N	N	N	N	N	N	Mild	N	N	NS
271	BN	61	M	884762	2	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
272	VM	28	M	882205	10	6	N	N	N	N	N	N	N	N	N	N	N	N	Severe	N	N	S
273	GS	22	M	884045	8	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
274	DS	24	M	873601	31	4	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
275	PK	50	F	877987	13	8	N	N	Y	N	N	N	N	N	Y	N	N	N	Severe	N	N	S
276	RK	65	M	927561	7	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
277	PK	50	F	928212	35	15	N	N	N	N	N	Y	N	N	N	N	N	N	Mild	N	N	S
278	SS	28	M	924184	24	6	N	N	Y	N	N	Y	N	N	N	N	N	N	Severe	N	N	S
279	KK	37	F	925034	19	11	N	N	Y	N	Y	N	N	N	N	N	N	N	Moderate	N	N	S
280	HS	60	M	928062	2	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
281	AS	50	M	924147	22	12	N	N	Y	N	N	N	N	N	N	N	N	N	Moderate	Y	N	NS
282	GS	38	M	890813	8	14	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
283	PK	64	M	922977	31	12	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
284	KB	38	M	916548	66	7	Y	Y	N	N	Y	Y	N	N	N	N	Y	N	Severe	N	N	NS
285	JK	48	F	924367	19	7	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
286	NS	21	M	736153	11	12	N	N	N	N	N	N	N	N	N	N	Y	N	Moderate	N	N	S
287	PK	60	F	745044	4	150	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
288	HS	26	M	729478	27	13	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
289	KK	59	F	565098	49	13	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
290	RS	32	M	722169	42	9	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
291	DS	26	M	747903	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
292	P	31	M	745913	5	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
293	RD	62	F	746627	7	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	Y	N	NS
294	S	50	M	710348	62	10	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	S
295	JS	70	M	745071	9	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
296	GS	30	M	745072	7	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
297	HK	38	F	749085	7	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
298	GK	50	F	743413	16	14	N	N	N	N	N	N	Y	N	N	N	N	N	Mild	N	N	S
299	JS	29	M	752056	4	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
300	KS	21	M	749150	9	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
301	TS	83	M	751980	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
302	AK	38	M	733411	36	13	N	N	N	N	N	N	N	N	N	N	N	N	Mild	Y	N	NS
303	GS	61	M	753637	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
304	NS	24	M	756339	1	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
305	NS	56	M	756539	2	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
306	RK	65	M	755208	4	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
307	RJ	18	M	749149	14	15	N	Y	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
308	MS	23	M	753463	7	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
309	NK	40	M	757980	14	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
310	BS	54	M	757934	1	11	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
311	JS	45	M	763549	5	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	S
312	HR	58	M	756601	19	10	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
313	PC	60	M	565005	7	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	Y	N	NS
314	R	44	M	814687	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS

S no.	Name (Initials)	Age (In years)	Sex	MRD number	Duration of stay (In days)	GCS	IVH	EDH	SDH	Cerebral hemoorhage	Hemmorahagic confusion	SAH	Intraparenchymal bleed	Axonal injury	Axial collection	Suligalical hemotoma	Fracture	Tentorium brush	Head Injury	Post traumatic seizure	Complication	Type of Management S/NS
315	M	40	M	704201	55	4	N	N	Y	N	N	N	N	N	N	N	N	N	Severe	N	N	S
316	NR	58	M	756677	18	12	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	S
317	KS	24	M	767067	1	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
318	I	54	M	4559	2	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
319	AT	19	M	757981	18	10	N	N	N	N	N	N	N	N	N	N	Y	N	Moderate	N	N	NS
320	A	30	M	743508	41	5	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
321	PK	30	F	767169	3	15	N	N	N	N	N	Y	N	N	N	N	N	N	Mild	Y	N	NS
322	RS	22	M	767030	6	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
323	AK	35	F	767040	6	12	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
324	SR	24	M	714803	88	4	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
325	RK	35	M	758089	25	8	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	Y	N	S
326	SK	22	M	767241	8	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	S
327	MS	55	M	766977	7	11	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
328	GS	45	M	767140	11	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
329	H	25	M	771757	6	10	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
330	KK	70	M	771768	6	15	N	N	Y	N		N	N	N	N	N	Y	N	Mild	N	N	NS
331	GS	21	M	772799	1	15	N	Y	N	N	Y	N	N	N	N	N	Y	N	Mild	N	N	NS
332	MK	50	F	782962	15	10	N	N	N	N	Y	Y	N	N	N	N	Y	N	Moderate	N	N	S
333	SR	24	M	714803	44	9	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	S
334	SS	31	M	793545	10	15	N	N	N	N	N	N	N	N	Y	N	N	N	Mild	N	N	NS
335	MS	45	M	798506	12	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
336	HS	28	M	802213	2	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
337	PS	28	M	793539	13	6	N	N	N	N	Y	N	N	N	N	N	Y	N	Severe	Y	N	NS
338	AS	49	M	802202	3	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
339	MS	35	M	800354	7	13	N	N	N	N	N	Y	N	N	N	N	Y	N	Moderate	N	N	NS
340	GS	45	M	779786	35	11	Y	N	Y	N	Y	N	N	N	N	N	Y	N	Mild	N	N	NS
341	KS	35	M	802244	6	14	N	N	N	N	Y	N	N	N	N	N	Y	N	Mild	N	N	S
342	S	65	F	805553	1	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
343	SS	31	M	799518	11	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
344	PS	25	M	805338	3	15	N	N	Y	N	Y	N	N	N	N	N	Y	N	Mild	N	N	NS
345	JS	29	M	800382	10	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
346	DB	26	M	805538	3	10	Y	N	N	N	Y	N	N	N	N	Y	N	N	Moderate	N	N	S
347	MS	21	M	776603	47	6	Y	N	N	N	Y	Y	N	N	N	N	Y	N	Severe	Y	N	S
348	BS	32	M	787442	32	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
349	HS	45	M	807344	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
350	SK	40	F	810451	5	15	N	N	N	N	Y	N	N	N	N	N	Y	N	Mild	N	N	NS
351	AS	22	M	807305	9	15	N	N	N	N	Y	N	N	N	N	N	Y	N	Mild	N	N	S
352	SK	27	F	783667	47	10	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
353	DS	27	M	877365	14	12	N	N	N	N	N	N	N	N	Y	N	N	N	Moderate	N	N	S
354	KS	40	M	870578	30	5	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
355	IG	60	F	883316	1	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
356	RS	55	M	877986	14	13	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	S
357	GS	62	M	882156	5	14	N	N	N	N	N	Y	N	N	N	N	N	N	Mild	Y	N	NS
358	BN	61	M	884762	2	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
359	VM	28	M	882205	10	6	N	N	N	N	N	N	N	N	N	N	N	N	Severe	N	N	S
360	GS	22	M	884045	8	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
361	DS	24	M	873601	31	4	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
362	PK	50	F	877987	13	8	N	N	Y	N	N	N	N	N	Y	N	N	N	Severe	N	N	S
363	RK	65	M	927561	7	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
364	PK	50	F	928212	35	15	N	N	N	N	N	Y	N	N	N	N	N	N	Mild	N	N	S
365	SS	28	M	924184	24	6	N	N	Y	N	N	Y	N	N	N	N	N	N	Severe	Y	N	S
366	KK	37	F	925034	19	11	N	N	Y	N	Y	N	N	N	N	N	N	N	Moderate	N	N	S

S no.	Name (Initials)	Age (In years)	Sex	MRD number	Duration of stay (In days)	GCS	IVH	EDH	SDH	Cerebral hemoorhage	Hemmorahagic confusion	SAH	Intraparenchymal bleed	Axonal injury	Axial collection	Suligalical hemotoma	Fracture	Tentorium brush	Head Injury	Post traumatic seizure	Complication	Type of Management S/NS
367	HS	60	M	928062	2	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
368	AS	50	M	924147	22	12	N	N	Y	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
369	GS	38	M	894521	8	14	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
370	PK	64	M	922977	31	12	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
371	KB	38	M	916548	66	7	Y	Y	N	N	Y	Y	N	N	N	N	Y	N	Severe	N	N	NS
372	JK	48	F	924367	19	7	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
373	HS	23	M	874962	13	7	N	Y	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
374	N	65	F	921242	41	7	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
375	KS	48	M	924768	11	7	Y	Y	Y	N	N	N	N	N	N	N	N	N	Severe	N	N	S
376	KR	57	M	924888	6	12	N	N	N	N	Y	Y	N	N	N	N	N	N	Mild	N	N	S
377	SS	22	M	781250	4	15	N	Y	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
378	M	24	M	698811	7	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	Y	N	NS
379	G	24	M	694812	16	7	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
380	S	41	M	672821	12	15	N	N	N	N	N	Y	N	N	N	N	Y	N	Mild	N	N	NS
381	B	72	M	701140	13	7	N	N	Y	N	Y	Y	N	N	N	N	N	N	Severe	N	N	NS
382	R	50	M	705048	9	15	N	N	N	Y	N	N	N	N	N	N	N	N	Mild	N	N	NS
383	H	32	M	707229	10	15	N	N	N	N	N	N	N	N	Y	N	N	N	Mild	N	N	NS
384	G	40	M	702111	16	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
385	AK	45	F	704411	17	15	N	N	Y	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
386	J	25	M	709503	11	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
387	PS	40	M	703658	14	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
388	BS	35	M	710453	3	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
389	VK	50	F	710255	7	15	N	N	N	N	Y	N	N	N	N	N	Y	N	Mild	Y	N	NS
390	S	30	M	679235	68	7	N	N	Y	N	Y	Y	N	N	N	N	Y	N	Severe	N	N	S
391	K	41	M	710354	4	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
392	P	41	F	707425	12	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
393	AS	60	M	741562	4	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
394	V	51	M	707870	14	11	N	Y	Y	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
395	N	17	F	761510	4	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
396	K	22	M	705840	13	12	N	N	Y	N	Y	Y	N	N	N	N	N	N	Mild	N	N	NS
397	H	70	M	696239	35	4	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
398	NP	34	F	688900	46	6	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
399	A	20	M	707259	27	6	N	N	Y	N	Y	Y	N	N	N	N	N	N	Severe	N	N	S
400	SS	35	M	761817	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	Y	N	NS
401	S	35	M	715773	4	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
402	D	50	M	710377	15	11	N	N	N	N	Y	N	N	N	N	N	N	N	Moderate	N	N	NS
403	RD	70	M	695924	42	7	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
404	MJ	38	M	720176	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
405	SS	70	M	703760	27	6	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	NS
406	SK	58	M	740339	5	12	N	N	Y	N		N	N	N	N	N	N	N	Moderate	N	N	NS
407	PS	25	M	716023	18	13	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
408	VK	65	M	713677	16	11	N	N	Y	N	N	N	N	N	N	N	N	N	Moderate	N	N	NS
409	PK	45	M	791812	16	12	N	N	N	N	N	N	Y	N	N	N	N	N	Moderate	Y	N	S
410	MS	28	M	739385	10	14	N	N	Y	N	N	Y	N	N	N	N	N	N	Moderate	N	N	NS
411	MS	26	M	722471	13	13	N	N	Y	N	Y		N	N	N	N	N	N	Mild	N	N	NS
412	PS	55	M	718845	18	10	N	Y	N	N	N	Y	N	N	N	N	N	N	Moderate	N	N	S
413	BK	46	M	729434	10	12	N	N	N	N	Y	Y	N	N	N	N	N	N	Moderate	N	N	NS
414	GS	36	M	718177	20	6	Y	N	N	N	N	N	N	N	N	N	Y	N	Severe	N	N	S
415	RS	48	M	720726	16	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
416	GS	26	M	724911	6	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
417	JK	50	M	698484	59	6	N	N	N	N	N	Y	N	N	N	N	N	N	Severe	N	N	S

S no.	Name (Initials)	Age (In years)	Sex	MRD number	Duration of stay (In days)	GCS	IVH	EDH	SDH	Cerebral hemoorhage	Hemmorahagic confusion	SAH	Intraparenchymal bleed	Axonal injury	Axial collection	Suligalical hemotoma	Fracture	Tentorium brush	Head Injury	Post traumatic seizure	Complication	Type of Management S/NS
418	RS	35	M	698828	52	3	N	Y	Y	N	Y	Y	N	N	N	N	N	N	Severe	N	N	S
419	RK	29	M	739364	16	7	N	N	Y	N	N	Y	N	N	N	N	N	N	Severe	N	N	S
420	DR	65	F	732797	6	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
421	Y	15	F	700318	8	8	N	Y	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
422	G	45	M	743310	5	15	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
423	KS	42	M	746396	4	14	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
424	A	25	M	728736	10	9	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	S
425	AR	54	M	727011	15	13	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
426	C	35	M	731554	10	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
427	S	18	M	677882	18	7	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
428	AK	27	M	733490	10	8	N	N	Y	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
429	J	21	M	736911	10	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	Y	N	S
430	KS	56	M	733162	15	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
431	R	26	F	734413	18	15	N	N	Y	N	N	N	N	N	N	N	N	N	MILD	N	N	S
432	HS	50	M	734581	13	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	S
433	A	29	M	740641	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
434	S	79	F	742168	5	14	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
435	NS	21	M	736215	11	12	N	N	N	N	N	N	N	N	N	N	Y	N	Moderate	N	N	S
436	PK	60	F	745144	4	150	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
437	HS	26	M	729878	27	13	N	N	Y	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
438	KK	59	F	569508	49	13	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	S
439	RS	32	M	722373	42	9	N	N	N	N	Y	N	N	N	N	N	N	N	Severe	N	N	S
440	DS	26	M	748703	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
441	P	31	M	745918	5	15	N	N	N	N	N	N	N	N	N	N	Y	N	Mild	N	N	NS
442	RD	62	F	746628	7	15	N	N	Y	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
443	S	50	M	710438	62	10	N	N	N	N	N	N	N	N	N	N	N	N	Moderate	N	N	S
444	JS	70	M	757548	9	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
445	GS	30	M	757529	7	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	S
446	HK	38	F	749185	7	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
447	GK	50	F	734316	16	14	N	N	N	N	N	N	Y	N	N	N	N	N	Mild	N	N	S
448	JS	29	M	752065	4	15	N	Y	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS
449	KS	21	M	749161	9	15	N	N	N	N	Y	N	N	N	N	N	N	N	Mild	N	N	NS
450	TS	83	M	752080	5	15	N	N	N	N	N	N	N	N	N	N	N	N	Mild	N	N	NS

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No. BFUHS/2K19p-TH/ 12723 **Date** 13/5/19

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.05.2019 has approved the plan of thesis of **Dr. Tarun Garg**, a student of your college for **MCh Neuro-Surgery** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis

Post traumatic seizure: a prospective study

Supervisor
Dr. Ashwani K. Chaudhary
Prof.,
Dept. of Neuro-Surgery,
DMCH, Ludhiana.

Co-Supervisor
Dr. Gagandeep Singh
Prof. & Head,
Dept. of Neurology,
DMCH, Ludhiana.

The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

POST TRAUMATIC SEIZURE: A PROSPECTIVE STUDY

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED
IN PARTIAL FULFILMENT OF THE REQUIREMENTS
FOR THE DEGREE OF

M.Ch.

In

NEUROSURGERY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

MARCH, 2019

DR. TARUN GARG

DEPARTMENT OF NEUROSURGERY
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	**POST TRAUMATIC SEIZURE : A PROSPECTIVE STUDY**
For the degree of	**M.Ch. Neurosurgery**
Name of the candidate	**Dr. Tarun Garg**
Supervisor	**Dr. Ashwani K Chaudhary,** Professor, Department of Neurosurgery, DMCH, Ludhiana
Co-supervisor	**Dr. Gagandeep Singh**, Professor and Head, Department of Neurology, DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

INTRODUCTION:TBI remains a significant public health problem. Individuals who survive moderate and severe TBI become at risk of developing secondary complications, including post-traumatic seizures (PTS). PTS are well-recognized sequelae of TBI. Despite previous research, there remains a high degree of variability in who will develop PTS and no approved prophylactic medications to prevent late PTS exist. Late PTS is associated with significant morbidity and worse outcomes following TBI.

AIMS & OBJECTIVES: The main objective of this study was to determine the incidence, time to event, and risk factors for posttraumatic seizures (PTS) following traumatic brain injury (TBI).

MATERIAL & METHODS: A prospective study shall be conducted on patients admitted with traumatic brain injury IN Dayanand Medical College and Hospital, Ludhiana from January 2019 to June 2020. Those part of the study shall have to satisfy the inclusion criteria and none of the exclusion criteria. All analyses were done using Statistical Package for Social Sciences Version 20 (SPSS Inc., Chicago, IL, USA) . P-values less than 0.05 were considered significant

CLINICAL SIGNIFICANCE: Human PTS results from a traumatic brain injury (TBI), which may result from traffic accident (~47%), fall (~30%), recreation or sport accident (14%), and assault (7%). The complexity and diversity of PTS parallels that of TBI, which includes both focal and diffuse components due to acceleration/deceleration injuries, blunt force closed head injuries with or without skull fracture, contusions, and penetrating brain injuries Therefore, it is of public health importance to understand the characteristics of individuals with PTS, identify factors to improve prognostication, and explore novel risk factors to support a personalized medicine approach.

KEYWORDS: POST TRAUMATIC SEIZURES (PTS), TRAUMATIC BRAIN INJURY (TBI).

Candidate's sign | Co- supervisor's sign | Supervisor's sign | Principal's sign

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"POST TRAUMATIC SEIZURE: A PROSPECTIVE STUDY"** exist in the Department of Neurosurgery, Dayanand Medical College, Ludhiana& will be provided to the candidate. We will see that the data being included in the thesis are genuine & is collected by the candidate himself under our supervision & guidance.

Supervisor

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Place: Ludhiana

Date: 27/4/2019

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	**Dr. Tarun Garg**
Department	**Neurosurgery**
Topic of Thesis	**POST TRAUMATIC SEIZURE : A PROSPECTIVE STUDY**
Likely date of appearing in PG examination	May 2021
Date of enrollment	25th August 2018
Name of Head of Department	Dr. R.K. Kaushal
1) Supervisor **2) Co-Supervisor**	Dr. Ashwani K Chaudhary Dr. Gagandeep Singh
Signature of Members of Research Committee withStamp	**Signature of Members of Ethics Committee with Stamp**
1. HOD Pharmacology	1. HOD Physiology
2. HOD Surgery	2. HOD Pathology
3. HOD Pathology	3. HOD Pharmacology
4. HOD Medicine	4. HODSPM
5. Dean Academics	5. HOD Medicine
6. Vice Principal	6. HOD Surgery
	7. HODObst. &Gynae
	8. Legal Experts
	9. Secretary, IEC
	10. Dean Academics
	11. Vice Principal
	12. Principal

Approved: Yes / No

Chairperson
Research Committee

Approved: Yes / ~~No~~

Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OFSUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr TARUN GARG H NO 55, PATEL NAGAR, BATHINDA 151001
2	NAME OF THE INSTITUTE	DAYANAND MEDICAL COLLEGE AND HOSPITAL, LUDHIANA
3	COURSE OF STUDY AND SUBJECT	M.CH. NEUROSURGERY
4	DATE OF ADMISSION OF COURSE	25 August, 2018
5	TITLE OF THE TOPIC	POST TRAUMATIC SEIZURE: A PROSPECTIVE STUDY

6 BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

TBI remains a significant public health problem. Individuals who survive moderate and severe TBI become at risk of developing secondary complications, including post-traumatic seizures (PTS). PTS are well-recognized sequelae of TBI. Despite previous research, there remains a high degree of variability in who will develop PTS and no approved prophylactic medications to prevent late PTS exist. Late PTS is associated with significant morbidity and worse outcomes following TBI.

Post-traumatic SEIZURE (PTE) refers to SEIZURE that develops after TBI.. Therefore, a common set of definitions adopted by many researchers is the following: (1) immediate seizures, usually defined as those occurring within 24 h after injury; (2) early seizures, which occur less than 1 week after injury; and (3) late seizures, which occur more than a week after injury. Since the risk of recurrence after a single late post-traumatic seizure is over 70%, most investigators consider a single late post-traumatic seizure as being sufficient for the diagnosis of PT

Human PTS results from a traumatic brain injury (TBI), which may result from traffic accident (~47%), fall (~30%), recreation or sport accident (14%), and assault (7%). The complexity and diversity of PTS parallels that of TBI, which includes both focal and diffuse components due to acceleration/deceleration injuries, blunt force closed head injuries with or without skull fracture, contusions, and penetrating brain injuries. Acceleration/deceleration injuries induce diffuse damage, while predominantly focal injuries induce surface

contusion and laceration that can be accompanied by skull fracture or hematoma

Therefore, it is of public health importance to understand the characteristics of individuals with PTS, identify factors to improve prognostication, and explore novel risk factors to support a personalized medicine approach.

6.2 REVIEW OF LITERATURE

As proposed by the International League Against Epilepsy and the International Bureau for Epilepsy in 2005, epilepsy is a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures, and by the neurobiologic, cognitive, psychological and social consequences of this condition.[1] Epilepsy is further defined as recurrent unprovoked seizures occuring at least 24 h apart.[2] Post-traumatic epilepsy (PTE) is a life-long complication of traumatic brain injury (TBI).[3]PTE and post-traumatic seizures (PTS) have both been used to describe seizures occurring after head trauma that are believed to be causally related to the trauma itself.[3] PTS are seizures occurring in the first week after TBI, and are considered to be provoked by head injury. PTE is defined as one or more unprovoked seizures that occur at least a week after TBI.[4] These are not universally accepted definitions, as some studies have allowed up to 4 weeks after head injury for early PTS.[3]

The occurrence of seizures after head injury is a recognized complication of TBI and has been demonstrated to worsen functional outcome significantly.[5] Bruns and Hauser performed an epidemiologic review of TBI and determined that the incidence of TBI is between 180 and 250 per 100,000 per year.[6] Other studies, using data from the CDC, report a higher incidence of TBI based on emergency room visits, hospitalizations and deaths.[7] The percentage of TBI patients who develop PTE is not known. It is estimated that TBI is an etiological factor in up to 20% of symptomatic epilepsies in the general population.[8]

In approximately half of PTE cases, seizures begin within the first year and in 80%, within the first 2 years. One population-based study estimated that 86%

of patients with one seizure, occurring at least 1 week after TBI, had a second seizure within 2 years.[9] A second population-based study reported the cumulative incidence of PTE as 9.1 per 100 persons in the first 3 years after TBI.[10] Variations in the estimation of the incidence of PTE are caused by differing definitions used for both TBI and PTE[3] and varying lengths of follow-up in studies. Most studies of TBI and PTE are not population based and may only include more severely injured individuals.[10]

The incidence of PTE in the civilian and military populations correlates with the severity of the inciting injury. This correlation between the incidence of PTE and severity of TBI explains most of the variability in the overall incidence of PTE, which ranges from 4 to 53%.[3,11,12] For example, in TBI with penetrating head injury, as with ballistic penetration, incidence of PTE was reported to be as high as 53%.[3,11] In 1980, Annegers*et al.* developed a three-tier classification for TBI severity: mild, moderate and severe (see Table 1).[13] A universally accepted classification for TBI severity has not yet been developed.[3]

Table 1

Classification of severity of traumatic brain injury.

Severity	Description
Mild TBI	No skull fracture found, and ≤30 min post-traumatic amnesia or LOC
Moderate TBI	Skull fracture or other injuries, post-traumatic amnesia or LOClasting between 30 min and 24 h, and not meeting criteria for severe TBI

Severity	Description
Severe TBI	Documented brain contusion, intracerebral hematoma, or >24 h post-traumatic amnesia or LOC

LOC: Loss of consciousness; TBI: Traumatic brain injury.[13]

The frequency of seizures in patients with PTE is not associated with any identifiable variables and often varies widely, even within generally homogeneous populations.[3] Several case studies and review articles have attempted to identify risk factors for PTE (Table 2). Upon reviewing the literature, Ferguson *et al.*[10] determined that there is an overall agreement that increased severity of TBI appears to lead to an increased risk of PTE.[9,14–18] The most consistent risk factor for PTS is the presence of intracerebral blood, which can result in up to a 30% increase in the risk of PTS.[3,19] The most consistently significant risk factor for PTE is the occurrence of early PTS (i.e., within 1 week after head injury).[3,20,21] The presence of subdural hematoma, brain contusion and multiple risk factors of severe TBI also increased the overall rate of PTE.[21] Increasing severity of PTE also correlated with higher seizure frequency, as well as epilepsy that is refractory to antiepileptic drug (AED) therapy.[20] Recently published data from the Vietnam Head Injury Study 35-year follow-up report suggested that patients with penetrating head injuries carry a high risk of developing PTE decades after their injury.[22] The same report also concluded lesion location, lesion size and lesion type were predictors of PTE.

Table 2

Independent risk factors for post-traumatic seizures and post-traumatic epilepsy.

Risk factors for PTS	Risk factors for PTE
Acute intracerebral hematoma	PTS
Acute subdural hematoma	Acute intracerebral hematoma
Younger age	Acute subdural hematoma
Increased injury severity	Brain contusion
Chronic alcoholism	Increased injury severity
	Age older than 65 years at time of injury

PTE: Post-traumatic epilepsy; PTS: Post-traumatic seizures.[3]

6.3 AIMS AND OBJECTIVES OF THE STUDY

To determine the frequency, time to event, and risk factors for posttraumatic seizures (PTS) following traumatic brain injury (TBI).

7.0 MATERIALS AND METHODS

7.1 SOURCE OF DATA

A prospective study shall be conducted on patients admitted with traumatic brain injury in Dayanand Medical College and Hospital, Ludhiana from 1st April 2019 to 31st May 2020.

7.2 METHODS OF COLLECTION OF DATA

Inclusion criterion

All patients who will present with acute traumatic brain injury in Dayanand medical college and hospital Ludhiana will be included in the study

Exclusion criterion

- A preceding history of brain injury or major mental disorder,
- Pregnancy,
- Predisposition to seizures (such as a history of brain tumor or stroke),
- Family history of seizure

In this study, we will use the classification of head injury severity as defined by Annegers et al. Mild injuries were those in which no skull fracture was found and the period of posttraumatic amnesia or loss of consciousness was ≤30 minutes. The moderately injured group of patients included those with skull fractures or other injuries with > 30 minutes of posttraumatic amnesia or loss of consciousness, who did not otherwise meet the criteria for a severe injury. Severe injuries were those with a documented brain contusion, an intracerebral hematoma (ICH) or > 24 hours of posttraumatic amnesia or loss of consciousness.[12]

Follow-up of 3 months will be done

Variables like age, sex, trauma severity, GCS, onset of PTS, and neuroradiological findings, number of antiepileptic medications will be studied. Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

Yes, Routine investigations, EEG, NC CT head

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached Herewith

8. LIST OF REFERENCES

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PROFORMA

S.No. __

Name	Age	Sex
Address	Ph. No.	
MRD No		
DOA	DOD	DOS

EXAMINATION

GCS at admission

Pupils

PR

BP

INVESTIGATIONS

NCCT HEAD FINDING

MRI BRAIN

Blood investigations

HB

TLC

PLATELETS

PT /INR

S.CREATININE

SERUM LFT

Signature of Candidate **Signature of Supervisor**

Selectrolytes

ECG

EEG

CALCIUM

MAGNESIUM

PHOSPHORUS

DAY	SEIZURE FREQUENCY
1(admission)	
7	
14	

HEAD INJURY CLASSIFICATION

MILD

MODERATE

SEVERE

ANTIEPILEPTIC-DOSE

FREQUENCY

Days of ICU Stay

Days of ventilator support

Type of management (surgical/ non surgical)

If surgical type of surgery

Complications

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____ निवासी _____ इस शोधकार्य **"POST TRAUMATIC SEIZURE : A PROSPECTIVE STUDY"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____ **"POST TRAUMATIC SEIZURE : A PROSPECTIVE STUDY"** ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂੰ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____
ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____

ਪਤਾ _____

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ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____

ਪਤਾ _____

_____
ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____
ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ